********** STN Columbus **********

FILE 'HOME' ENTERED AT 11:51:10 ON 31 JAN 97

=> file medline scisearch embase biosis
COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 0.15 0.15

=> s nasal or intranasal
TOTAL FOR ALL FILES
L5 112227 NASAL OR INTRANASAL

=> s l5 and (administration or immunization)
TOTAL FOR ALL FILES
L10 19823 L5 AND (ADMINISTRATION OR IMMUNIZATION)

=> s l10 and antibod?
TOTAL FOR ALL FILES
L15 2670 L10 AND ANTIBOD?

=> s l15 and pathogen?
TOTAL FOR ALL FILES
L20 263 L15 AND PATHOGEN?

=> dup rem l20
PROCESSING COMPLETED FOR L20
L21 197 DUP REM L20 (66 DUPLICATES REMOVED)

=> s l21 and((antigen presenting cell# or APC#)or(b(W)(cell# or lymphocyte#))or(t(W)(cell# or lymphocytes)))
L22 157 S L21
L23 13 FILE MEDLINE
L24 13 S L21
L25 3 FILE SCISEARCH
L26 22 S L21
L27 3 FILE EMBASE
L28 5 S L21
L29 0 FILE BIOSIS

TOTAL FOR ALL FILES
L30 19 L21 AND((ANTIGEN PRESENTING CELL# OR APC#) OR(B(W)(CELL# OR LYMPHOCYTE#)) OR(T(W)(CELL# OR LYMPHOCYTES)))

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L30 ANSWER 1 OF 19 MEDLINE
ACCESSION NUMBER: 96351449 MEDLINE
TITLE: Manipulating systemic and mucosal immune responses with skin-deliverable adjuvants.
AUTHOR: Chin J; San Gil F; Novak M; Eamens G; Djordjevic S; Simecka J; Duncan J; Mullbacher A
CORPORATE SOURCE: NSW Agriculture, Elizabeth Macarthur Agricultural Institute, Sydney, Australia.
SOURCE: JOURNAL OF BIOTECHNOLOGY, (1996 Jan 26) 44 (1-3) 13-9. Ref: 12
Journal code: AL6. ISSN: 0168-1656.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals; B
ENTRY MONTH: 9612
ABSTRACT:
Most medically important bacterial and viral ***pathogens*** gain entry into the body either via the skin or a mucosal surface. Vaccination provides a viable and cost-effective strategy for the prevention of such diseases and it has always been a principal aim with vaccinologists, to be able to promote simultaneously, protective immune responses both systemically and at mucosal surfaces. The paradigm that mucosal immunity is best stimulated by exposure to antigen via a mucosal route simply because inductive sites such as Peyer's patches and bronchial associated lymphoid tissues are located in the mucosal epithelium, has promoted a plethora of immunizing strategies aimed at delivering both antigen and adjuvant to mucosal surfaces. We have developed a novel adjuvant system capable of intradermal delivery of antigens complexed in an ISCOSOME delivery vehicle. This adjuvant, referred to as a skin and mucosal adjuvant or SAMA4, was efficacious in eliciting both systemic and mucosal IgG and IgA ***antibodies*** in sheep, pigs and mice. SAMA4 does not induce granulomatous lesions at the site of vaccine delivery and can be used to deliver adjuvanted antigens by other routes including ***intranasal*** , oral and intravaginal. Using ovalbumin as a test antigen, intradermally delivered ovalbumin-SAMA4 complexes was found to be very effective in promoting a cytotoxic ***T*** ***cell*** response. Attempts to dissect the mode of action of SAMA4 by flow cytometric analysis of lymphocyte populations from the spleen, lung, liver and thymus revealed an effect of route of vaccine delivery upon the composition of specific lymphocyte subsets in these various organ compartments. From this, it can be inferred that SAMA4 induced a route-dependent re-mobilization and alteration in lymphocyte trafficking patterns. Other mucosal adjuvants such as cholera toxin B and microspheres, when injected intradermally, tended to promote primarily, an IgG and not an IgA response against hte carrier antigen.

CONTROLLED TERM: Check Tags: Animal; Comparative Study; Female; Human; Support, Non-U.S. Gov't
*** Antibody Formation***
****Bacterial Vaccines: AD, administration &***
*** dosage***
Bacterial Vaccines: IM, immunology
Injections, Intradermal
*Intestinal Mucosa: IM, immunology
Lung: IM, immunology
Mice
Peyer's Patches: IM, immunology
Sheep
Swine
Vaccination: MT, methods
****Viral Vaccines: AD, administration & dosage***
Viral Vaccines: IM, immunology
CHEMICAL NAME: 0 (Bacterial Vaccines); 0 (Viral Vaccines)

L30 ANSWER 2 OF 19 MEDLINE
ACCESSION NUMBER: 96264828 MEDLINE
TITLE: ***Immunization*** with a soluble recombinant HIV protein entrapped in biodegradable microparticles induces HIV-specific CD8+ cytotoxic ***T*** ***lymphocytes*** and CD4+ Th1 cells.
AUTHOR: Moore A; McGuirk P; Adams S; Jones W C; McGee J P; O'Hagan D T; Mills K H
CORPORATE SOURCE: Biology Department, St. Patrick's College, Maynooth, Co. Kildare, Ireland.
SOURCE: VACCINE, (1995 Dec) 13 (18) 1741-9.
Journal code: X6O. ISSN: 0264-410X.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 9611
ABSTRACT:
One of the major obstacles to the development of successful recombinant vaccines against human immunodeficiency virus (HIV) and other intracellular ***pathogens*** is the identification of a safe and effective vaccine delivery system for the induction of cell mediated immunity with soluble protein antigens. In this study it was demonstrated that ***immunization*** with a recombinant HIV envelop (env) protein entrapped in biodegradable poly(lactide-co-glycolide) (PLG) microparticles induced consistent HIV-specific CD4+ and CD8+ ***T*** - ***cell*** responses in mice. Major histocompatibility complex (MHC) class I-restricted cytotoxic ***T*** ***lymphocytes*** (CTL) responses were detected following a single systemic ***immunization*** with gp120 entrapped microparticles and when given by the ***intranasal*** (i.n.) route induced HIV-specific CD8+ CTL and secretory IgA. Furthermore ***immunization*** with gp120 entrapped in microparticles generated CD4+ ***T*** ***cells*** that secreted moderate to high levels of IFN-gamma. Therefore, PLG microparticles are a safe and effective means of delivering antigen to the appropriate processing site for the generation of class I-restricted CTL, and are also capable of inducing Th1 cells.

CONTROLLED TERM: Check Tags: Animal; Support, Non-U.S. Gov't
*** Antibody Specificity***
Biocompatible Materials
Biodegradation
Cell Division: IM, immunology
Cytotoxicity, Immunologic
****CD8-Positive T-Lymphocytes: IM, immunology***
Drug Carriers
*** HIV Envelope Protein gp120: AD, administration*** & dosage***
*HIV Envelope Protein gp120: IM, immunology
****Immunization***
Mice
Mice, Inbred BALB C
Microspheres
Polymers
Solubility
*Th1 Cells: IM, immunology
CHEMICAL NAME: 0 (polylactic acid-polyglycolic acid copolymer); 0 (Biocompatible Materials); 0 (Drug Carriers); 0 (HIV Envelope Protein gp120); 0 (Polymers)

L30 ANSWER 3 OF 19 MEDLINE
ACCESSION NUMBER: 96259573 MEDLINE
TITLE: Oral tolerance: mechanisms and possible role in inflammatory joint diseases.
AUTHOR: Kagnoff M F
CORPORATE SOURCE: Laboratory of Mucosal Immunology, University of California, San Diego, La Jolla 92093-0623, USA.
CONTRACT NUMBER: DK35108 (NIDDK)
DK47739 (NIDDK)
SOURCE: BAILLIERES CLINICAL RHEUMATOLOGY, (1996 Feb) 10 (1) 41-54. Ref: 71
Journal code: CRY. ISSN: 0950-3579.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 9610
ABSTRACT:
Decreased systemic immune responsiveness to a specific antigen following exposure to that antigen by the enteric route is termed 'oral tolerance.' Oral tolerance is revealed when attempts are made to parenterally immunize the host to the same antigen that was previously administered orally or intragastrically. A similar phenomenon is also seen following antigen exposure via the ***nasal*** mucosa and a related phenomenon is seen following antigen exposure in the upper respiratory tract. There has been a marked renewal of interest in the mechanisms that underlie oral tolerance because of its potential role for preventing and treating autoimmune and inflammatory diseases and IgE-mediated allergic disorders. The specific factors that determine whether or not the host develops mucosal tolerance to an antigen administered by the mucosal route are also of substantial importance for those involved in mucosal vaccine development. Furthermore, putative abnormalities in the ability of the host to develop mucosal tolerance may play a ***pathogenetic*** role in certain autoimmune and allergic diseases and disorders. Several well-defined immunological mechanisms mediate oral tolerance. These include the induction, following mucosal antigen exposure, of regulatory populations of ***T*** - ***cells*** that can down-regulate specific immune responses (e.g. DTH) via the production of specific cytokines (e.g. TGF-beta 1, IL-10 and IL-4). In addition, clonal anergy, clonal deletion and ***antibody*** -mediated suppression can be shown to play a role in the induction and maintenance of mucosal tolerance in several experimental systems. In animal studies, the onset of collagen-induced, adjuvant-induced, antigen-induced and pristane-induced arthritis has been delayed and the severity of ongoing disease diminished following feeding collagen type II. Mucosal tolerance has been clearly demonstrated in humans and clinical studies have been undertaken to treat rheumatoid arthritis using a similar approach. Results of initial clinical studies in rheumatoid arthritis indicated a modest improvement and further studies are ongoing in this and other autoimmune diseases

(e.g. multiple sclerosis, autoimmune uveitis and insulin-dependent diabetes). This approach, if successful, could offer a new and novel therapeutic modality for preventing autoimmune and allergic disorders, and modulating ongoing disease.

CONTROLLED TERM: Check Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
*** Administration, Oral***
****Antigens: AD, administration & dosage***
*Antigens: IM, immunology
*Arthritis, Rheumatoid: IM, immunology
Arthritis, Rheumatoid: TH, therapy
Disease Models, Animal
*Immune Tolerance
Mucous Membrane: IM, immunology
CHEMICAL NAME: 0 (Antigens)

L30 ANSWER 4 OF 19 MEDLINE
ACCESSION NUMBER: 96066237 MEDLINE
TITLE: BHV-1 glycoprotein I and recombinant interleukin 1 beta efficiently elicit mucosal IgA response.
AUTHOR: Gao Y; Daley M J; Splitter G A
CORPORATE SOURCE: Department of Animal Health and Biomedical Sciences, University of Wisconsin-Madison 53706, USA..
SOURCE: VACCINE, (1995 Jun) 13 (9) 871-7.
Journal code: X6O. ISSN: 0264-410X.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 9602
ABSTRACT:
The mucosal immune response to most soluble antigens administered directly to the mucosal system is low and requires a large amount of antigen and frequent vaccinations. In this study we tested whether immunizing cattle at a site which shares lymphatic drainage with the ***nasal*** mucosa could prime local mucosal immunity. We further tested whether recombinant bovine IL-1 beta (rBoIL-1 beta) could potentiate the induction of mucosal immunity. Animals were immunized subcutaneously at the base of the ear (s.e.) with recombinant bovine herpesvirus-1 (BHV-1) envelope glycoprotein I (gI) (35 micrograms animal-1) emulsified in incomplete Freund's adjuvant with or without rBoIL-1 beta (500 ng kg-1) followed by a second ***immunization*** 42 days later. Animals were challenged with virulent BHV-1 intranasally 42 days after the second ***immunization*** . Mucosal IgA from the nares was induced after only one ***immunization*** , and enhanced by boosting. rBoIL-1 beta treated animals had higher levels of BHV-1 specific ***nasal*** IgA ($p < 0.01$) and serum neutralizing ***antibody*** ($p < 0.05$). rBoIL-1 beta-treated animals also had increased numbers of surface IgA+ ($p < 0.05$) and IgG1+ ($p < 0.001$) ***B*** ***cells*** after in vitro antigen (gI) stimulation of peripheral blood lymphocytes suggesting that there was a greater expension of IgA+ and IgG1+ ***B*** ***cells*** in rBoIL-1 beta treated animals. When challenged with BHV-1, 3 of 4 animals in the gI+rBoIL-1 beta group were fully protected from viral replication in the nares, while only 1 of 4 animals receiving gI alone was protected.(ABSTRACT TRUNCATED AT 250 WORDS)

CONTROLLED TERM: Check Tags: Animal; Male; Support, U.S. Gov't, Non-P.H.S.
*** Antibodies, Viral: BI, biosynthesis***
Cattle
Cattle Diseases: IM, immunology
Cattle Diseases: PC, prevention & control
Cell Division: IM, immunology
Cell Line
Herpesviridae Infections: IM, immunology
Herpesviridae Infections: PC, prevention & control
*Herpesvirus 1, Bovine: IM, immunology
*** Herpesvirus 1, Bovine: PY, pathogenicity***
IgA: BI, biosynthesis
*IgA: IM, immunology
*Interleukin-1: IM, immunology
Monocytes: CY, cytology
****Nasal Mucosa: IM, immunology***
Neutralization Tests
Recombinant Proteins: IM, immunology
Respiratory Tract Diseases: IM, immunology
Respiratory Tract Diseases: PC, prevention & control
Vaccines, Synthetic: IM, immunology
*Viral Proteins: IM, immunology
Virus Replication: IM, immunology
CHEMICAL NAME: 0 (bovine herpesvirus type-1 glycoproteins); 0 (***Antibodies*** , Viral); 0 (IgA); 0 (Interleukin-1); 0 (Recombinant Proteins); 0 (Vaccines, Synthetic); 0 (Viral Proteins)

L30 ANSWER 5 OF 19 MEDLINE
ACCESSION NUMBER: 95273775 MEDLINE
TITLE: ***Pathogenicity*** of neutralization escape mutants of mouse hepatitis virus: correlation with T- and ***B*** - ***cell*** depletions.
AUTHOR: Lamontagne L; Page C; Braunwald J; Martin J P
CORPORATE SOURCE: Departement des Sciences Biologiques, Universite du Quebec `a Montreal, Que., Canada..
SOURCE: RESEARCH IN IMMUNOLOGY, (1994 Sep) 145 (7) 553-65.
Journal code: R6E. ISSN: 0923-2494.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 9508
ABSTRACT:
Viral ***pathogenicity*** is a result of an imbalance between viral replication and the host's immune defences. When the virus is lymphotropic, understanding the ***pathogenic*** process of the viral disease becomes complicated because virus/lymphocyte interactions can alter the cell's integrity and subsequently induce immunodeficiency. The immune system plays an important role in the outcome of acute disease induced by the mouse hepatitis virus type 3 (MHV3). The use of attenuated escape mutants provides a tool to study the role of viral properties involved in its ***pathogenicity*** . We selected MHV3 mutants by

virtue of their resistance to neutralization by monoclonal ***antibodies*** (mAb), in order to study their ***pathogenic*** properties. We reported that two MHV3 escape mutants were attenuated in their ***pathogenic*** properties according to inoculation site and with regard to survival time and ability to deplete T- and ***B*** - ***cell*** subpopulations in the spleen, thymus and bone marrow of susceptible Balb/c mice. The highly attenuated CL12 mutant could not induce depletion in T or ***B*** ***cells*** following *intraperitoneal* (i.p.) or ***intranasal*** (i.n.) inoculations, at three days postinfection. The less attenuated 51.6 mutant, however, maintained the ability to deplete T *and* ***B*** ***cells*** following i.p. inoculation, as described with the ***pathogenic*** MHV3. In contrast, no depletion of ***T*** ***cells*** following i.n. inoculation was induced with this mutant, although B lineage cells decreased. The use of such mutants enabled us to examine the role of each compartment of the immune system, since the highly attenuated CL12 mutant induced no immunodeficiency, as defined by immune cell depletion, whereas the less attenuated 51.6 mutant maintained its ability to decrease only the ***B*** - ***cell*** compartment after i.n. inoculation. Results are discussed with regard to the virus/lymphocyte interactions during the ***pathogenic*** process.

CONTROLLED TERM: Check Tags: Animal; Comparative Study; Support, Non-U.S. Gov't
*** Administration, Intranasal***
****Antibodies, Monoclonal: IM, immunology***
****Antibodies, Viral: IM, immunology***
****B-Lymphocyte Subsets: IM, immunology***
Brain: PA, pathology
Brain: VI, virology
*Coronavirus Infections: VI, virology
Gastroenteritis Virus, Murine: GE, genetics
Gastroenteritis Virus, Murine: IM, immunology
Gastroenteritis Virus, Murine: PH, physiology
****Gastroenteritis Virus, Murine: PY,*** *** pathogenicity***
*Hepatitis, Viral, Animal: VI, virology
Injections, Intraperitoneal
L Cells
*Lymphocyte Depletion
Lymphoid Tissue: PA, pathology
Lymphoid Tissue: VI, virology
Mice
Mice, Inbred BALB C
Neutralization Tests
*T-Lymphocyte Subsets: IM, immunology
Virulence: GE, genetics
Virus Replication
Viscera: PA, pathology
Viscera: VI, virology
CHEMICAL NAME: 0 (***Antibodies*** , Monoclonal); 0 (***Antibodies*** , Viral)

L30 ANSWER 6 OF 19 MEDLINE
ACCESSION NUMBER: 95247285 MEDLINE
TITLE: ***Antibody*** and cytokine responses in a mouse pulmonary model of Shigella flexneri serotype 2a infection.
AUTHOR: van de Verg L L; Mallett C P; Collins H H; Larsen T; Hammack C; Hale T L
CORPORATE SOURCE: Department of Enteric Infections, Walter Reed Army Institute of Research, Washington, D.C. 20307, USA..
SOURCE: INFECTION AND IMMUNITY, (1995 May) 63 (5) 1947-54. Journal code: GO7. ISSN: 0019-9567.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 9508
ABSTRACT:
A murine pulmonary model was used to study the mucosal immune response to Shigella flexneri serotype 2a infection. Inoculation of BALB/cJ mice with shigellae via the ***intranasal*** route resulted in bacterial invasion of bronchial and alveolar epithelia with concomitant development of acute suppurative bronchiolitis and subsequent development of lethal pneumonia. The pathology of pulmonary lesions resembled the colitis that characterizes shigellosis in humans and primates. Significant protection against a lethal dose of S. flexneri 2a was observed in mice previously infected with two sublethal doses of the homologous strain. Immunity against lethal challenge was associated with decreased bacterial invasion of the mucosal epithelium. Over the course of two sublethal challenges, which constituted primary and secondary immunizations, mice developed pulmonary and serum immunoglobulin G and A ***antibody*** recognizing both lipopolysaccharide and invasion plasmid antigens IpaB and IpaC. Immune mice and naive control mice differed in lung lavage cytokine levels following lethal challenge. Immune mice developed significantly elevated levels of pulmonary gamma interferon within 6 h of challenge, while naive control mice developed elevated levels of this cytokine later during the initial 24-h period. Both groups had elevated levels of gamma interferon during the 24- to 48-h period of infection. Both groups also had elevated levels of tumor necrosis factor alpha within 6 h of challenge, but the control mice had significantly higher levels at the 48- and 72-h time points. Elevated levels of interleukin-4 were observed only in immunized mice. This cytokine appeared within 24 h and receded between 48 and 72 h. Fluorescence-activated cell sorter analysis of lung parenchymal cells showed that both groups experienced an initial influx of monocytes, but the proportion of this cell type began to recede in immunized mice after 48 h of infection, while peak levels were maintained in the control animals. These studies suggest that elements of local ***B*** ***lymphocyte*** activity, as well as Th1 and Th2 lymphocyte activity, may contribute to the survival of immune mice after ***intranasal*** challenge with shigellae.

CONTROLLED TERM: Check Tags: Animal; Female
*** Administration, Intranasal***
****Antibodies, Bacterial: BI, biosynthesis***
Bacterial Proteins: IM, immunology
Bronchiolitis
*Cytokines: BI, biosynthesis
Disease Models, Animal
*Dysentery, Bacillary: IM, immunology
Dysentery, Bacillary: MO, mortality

Dysentery, Bacillary: PC, prevention & control
Enzyme-Linked Immunosorbent Assay
Flow Cytometry
*** Immunization***
Immunoblotting
Lung: IM, immunology
Lung: MI, microbiology
Lung: PA, pathology
Mice
Mice, Inbred BALB C
Mucous Membrane: IM, immunology
*Pneumonia, Bacterial: IM, immunology
Pneumonia, Bacterial: MO, mortality
Pneumonia, Bacterial: PC, prevention & control
Serotyping
Shigella flexneri: CL, classification
*Shigella flexneri: IM, immunology
*** Shigella flexneri: PY, pathogenicity***
Survival Analysis
CHEMICAL NAME: 0 (ipaB protein); 0 (***Antibodies*** , Bacterial); 0 (Bacterial Proteins); 0 (Cytokines)

L30 ANSWER 7 OF 19 MEDLINE
ACCESSION NUMBER: 95040349 MEDLINE
TITLE: ***T*** ***cell*** -derived antigen binding molecules play a role in the induction of airway hyperresponsiveness.
AUTHOR: Garssen J; Nijkamp F P; Van Vugt E; Van der Vliet H; Van Loveren H
CORPORATE SOURCE: National Institute of Public Health and Environmental Protection, Bilthoven, The Netherlands..
SOURCE: AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE, (1994 Dec) 150 (6 Pt 1) 1528-38.
Journal code: BZS. ISSN: 1073-449X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 9502
ABSTRACT:
We previously demonstrated that tracheal hyperreactivity (in vitro) and altered lung functions (in vivo) were induced during a delayed-type hypersensitivity (DTH) reaction in murine lungs. These alterations were transferable with ***T*** ***cells*** , suggesting that this animal model could be used as a model for cellular IgE-independent immunity. In the present study we demonstrated that depletion of T suppressor/cytotoxic cells failed to abolish the ability of transferred cells to induce hyperresponsiveness. Depletion of T helper cells partially inhibited the induction of hyperreactivity. Depletion of 14-30+ cells (the monoclonal ***antibody*** 14-30 reacts with a common isotype of ***T*** ***cell*** -derived antigen binding molecules [TABM] that can arm mast cells) completely abolished the ability to transfer hyperreactivity. The cromoglycate-like antiasthmatic drug nedocromil, which stabilizes mast cells, inhibited the induction of ***T*** ***cell*** -mediated hyperresponsiveness. Moreover, in mast cell-deficient mice, ***T*** ***cell*** -mediated hyperresponsiveness can be less induced compared with normal littermates. These experiments indicate that mast cells play at least a partial role in the induction of airway hyperresponsiveness in this model. Dexamethasone, a well-known inhibitor of phospholipase A2, inhibited the ***T*** ***cell*** -mediated hyperresponsiveness, whereas the cyclooxygenase inhibitor suprofen did not. This indicated that arachidonic acid metabolites, but not cyclooxygenase products, play a role in the induction of ***T*** ***cell*** -mediated hyperreactivity. Pretreatment with the lipoxygenase inhibitor AA-861 significantly inhibited the induction of tracheal hyperreactivity. Platelet-activating factor appeared not to be involved in the induction of hyperresponsiveness in this model, because the platelet-activating factor antagonist WEB 2170 failed to abolish the induction of ***T*** ***cell*** -mediated hyperreactivity. Intravenous injection of purified mast cell-arming TABM, followed by ***intranasal*** hapten challenge 30 min later, resulted in increased vascular permeability 2 h after challenge, which is characteristic of the early initiating phase of DTH. In addition, tracheal hyperreactivity (in vitro) and altered lung functions (in vivo) were observed 2 h after challenge. From these data we conclude that airway hyperreactivity and altered lung functions are induced by early steps in the cellular cascade of DTH.

CONTROLLED TERM: Check Tags: Animal; Comparative Study; Male; Support, Non-U.S. Gov't
Airway Resistance: DE, drug effects
Airway Resistance: IM, immunology
*Bronchial Hyperreactivity: ET, etiology
Bronchial Hyperreactivity: IM, immunology
Bronchial Hyperreactivity: PP, physiopathology
Capillary Permeability: DE, drug effects
Capillary Permeability: IM, immunology
Hypersensitivity, Delayed: ET, etiology
Hypersensitivity, Delayed: IM, immunology
Hypersensitivity, Delayed: PP, physiopathology
*** Immunization, Passive: MT, methods***
Lung: DE, drug effects
Lung: PP, physiopathology
Mast Cells: DE, drug effects
Mast Cells: IM, immunology
Mice
Mice, Inbred BALB C
*Picryl Chloride: PD, pharmacology
*** Receptors, Antigen, T-Cell: DE, drug effects***
****Receptors, Antigen, T-Cell: IM, immunology***
*** Specific Pathogen-Free Organisms***
*** T-Lymphocytes: DE, drug effects***
****T-Lymphocytes: IM, immunology***
Trachea: DE, drug effects
Trachea: IM, immunology
Trachea: PP, physiopathology
Vaccination: MT, methods
CAS REGISTRY NO.: 88-88-0 (Picryl Chloride)
CHEMICAL NAME: 0 (Receptors, Antigen, ***T*** - ***Cell***)

L30 ANSWER 8 OF 19 MEDLINE
ACCESSION NUMBER: 95038296 MEDLINE

TITLE: Exploration of mucosal immunity in humans: relevance to vaccine development.
AUTHOR: Czerkinsky C; Holmgren J
CORPORATE SOURCE: INSERM Unit 80, Hopital Edouard-Herriot, Lyon, France..
CONTRACT NUMBER: 3RO1HD26634-0151 (NICHD)
SOURCE: CELLULAR AND MOLECULAR BIOLOGY, (1994) 40 Suppl 1 37-44. Ref: 21
Journal code: BNA.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 9502
ABSTRACT:
Although the immune system is remarkably diverse, there is evidence that certain types of immune responses take place and are restricted to certain anatomic locations within the body. The concept of a common mucosal immune system that provides immune reactivity not only at the site of antigen deposition but also at remote mucosal sites may be explained by the utilization of organ-specific recognition molecules by circulating precursors of mucosal immunoblasts and by the production of certain maturation factors (e.g. cytokines, hormones) produced preferentially in certain organs or parts of a given organ. This notion may explain the unification of immune responses in diverse mucosal sites and the physiologic segregation of mucosal from systemic immune mechanisms. Novel methods have been developed to enable studies of antigen specific B and ***T*** ***cell*** responses in various mucosal and extramucosal tissues in primates and rodents, using cholera toxin or its B subunit as prototype immunogens and mucosal carrier-delivery systems. The tissue localization and isotype commitment of ***antibody*** -secreting cells (ASC) and the homing potential of their circulating precursors have also been examined after oral, ***nasal*** , intra-tonsillar, rectal and/or genital ***immunization*** (s). The anatomical distribution of T- and accessory cell-derived cytokines has also been examined. These tools and approaches are being employed in studies attempting to induce optimal mucosal immune responses to several mucosal ***pathogens*** including HIV-1, in certain organs such as the lower gastrointestinal tract and the female urogenital tract.(ABSTRACT TRUNCATED AT 250 WORDS)

CONTROLLED TERM: Check Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
*** Antibody-Producing Cells: IM, immunology***
*** Cholera Toxin: AD, administration & dosage***
Cholera Toxin: IM, immunology
Escherichia coli: IM, immunology
Gastric Mucosa: IM, immunology
Immunity
Intestinal Mucosa: IM, immunology
*Mucous Membrane: IM, immunology
Nasopharynx: IM, immunology
Primates
*Vaccines: IP, isolation & purification
CAS REGISTRY NO.: 9012-63-9 (Cholera Toxin)
CHEMICAL NAME: 0 (Vaccines)

L30 ANSWER 9 OF 19 MEDLINE
ACCESSION NUMBER: 94095926 MEDLINE
TITLE: Development of the airway intraepithelial dendritic cell network in the rat from class II major histocompatibility (Ia)-negative precursors: differential regulation of Ia expression at different levels of the respiratory tract.
AUTHOR: Nelson D J; McMenamin C; McWilliam A S; Brenan M; Holt P G
CORPORATE SOURCE: Division of Cell Biology, Western Australian Research Institute for Child Health, Princess Margaret Hospital, Subiaco..
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1994 Jan 1) 179 (1) 203-12.
Journal code: I2V. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 9404
ABSTRACT:
The relative inefficiency of respiratory mucosal immune function during infancy is generally attributed to the immaturity of the neonatal ***T*** ***cell*** system. However, immune competence in the adult lung has recently been shown to be closely linked to the functional capacity of local networks of intraepithelial dendritic cells (DC). This study examines the density and distribution of these DC throughout the neonatal respiratory tract in rats, focusing particularly on microenvironmental regulation of their class II major histocompatibility complex (MHC) (Ia) expression. In animals housed under dust-controlled conditions, airway epithelial and alveolar Ia+ DC detectable by immunostaining with the monoclonal ***antibody*** (mAb) Ox6 are usually not seen until day 2-3 after birth, and adult-equivalent staining patterns are not observed until after weaning. In contrast, the mAb Ox62 detects large numbers of DC in fetal, infant, and adult rat airway epithelium. Costaining of these Ox62+ DC with Ox6 is rare in the neonate and increases progressively throughout infancy, and by weaning Ia+ DC comprised, on average, 65% of the overall intraepithelial DC population. In infant rats, Ia+ DC are observed first at the base of the ***nasal*** turbinates, sites of maximum exposure to inhaled particulates, suggesting that their maturation is driven in part by inflammatory stimuli. Consistent with this suggestion, densitometric analysis of Ia staining intensity of individual DC demonstrates that by 2-3 d after birth, Ia expression by ***nasal*** epithelial DC was comparable with that of Iahigh epidermal Langerhans cells in adjacent facial skin, at a time when expression by tracheal epithelial DC was 7-10-fold lower. Additionally, the rate of postnatal appearance of Iahigh DC in the airway epithelium was increased by ***administration*** of interferon gamma, and decreased by exposure of infant rats to aerosolized steroid. These findings collectively suggest that Ia expression by neonatal respiratory tract DC is locally controlled and can be upregulated by mediators that are produced within the lung and airway epithelium in response to inhalation of proinflammatory stimuli. It was

also noted that lalow neonatal airway DC expressed adult equivalent levels of class I MHC, which suggests differences in capacity to prime for CD8(+)-dependent versus CD4(+)-dependent immunity to inhaled ***pathogens*** , during the early postnatal period.

CONTROLLED TERM: Check Tags: Animal; Support, Non-U.S. Gov't
Androstadienes: PD, pharmacology
Animals, Newborn
Anti-Inflammatory Agents, Steroidal: PD, pharmacology
*Dendritic Cells: CY, cytology
Dendritic Cells: IM, immunology
Epithelium: CY, cytology
Epithelium: DE, drug effects
Epithelium: GD, growth & development
Epithelium: IM, immunology
Flow Cytometry
*Histocompatibility Antigens Class II: BI, biosynthesis
Histocompatibility Antigens Class II: IM, immunology
Rats
Respiratory System: CY, cytology
*Respiratory System: IM, immunology
*Trachea: CY, cytology
Trachea: DE, drug effects
Trachea: GD, growth & development
Trachea: IM, immunology
CAS REGISTRY NO.: 80474-14-2 (fluticasone)
CHEMICAL NAME: 0 (Androstadienes); 0 (Anti-Inflammatory Agents, Steroidal); 0 (Histocompatibility Antigens Class II)

L30 ANSWER 10 OF 19 MEDLINE
ACCESSION NUMBER: 93059699 MEDLINE
TITLE: Pulmonary histopathology induced by respiratory syncytial virus (RSV) challenge of formalin-inactivated RSV-immunized BALB/c mice is abrogated by depletion of CD4+ ***T*** ***cells*** .
AUTHOR: Connors M; Kulkarni A B; Firestone C Y; Holmes K L; Morse H C 3d; Sotnikov A V; Murphy B R
CORPORATE SOURCE: Respiratory Viruses Section, National Institute of Allergy and Infectious Diseases, Bethesda, Maryland 20892..
SOURCE: JOURNAL OF VIROLOGY, (1992 Dec) 66 (12) 7444-51. Journal code: KCV. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 9302
ABSTRACT:
In previous studies, it was observed that children immunized with a formalin-inactivated respiratory syncytial virus vaccine (FI-RSV) developed severe pulmonary disease with greater frequency during subsequent natural RSV infection than did controls. During earlier efforts to develop an animal model of this phenomenon, enhanced pulmonary histopathology was observed after ***intranasal*** RSV challenge of FI-RSV-immunized cotton rats. Progress in understanding the immunologic basis for these observations has been hampered by the lack of reagents useful in manipulating the immune response of the cotton rat. This problem prompted us to reinvestigate the characteristics of immunity to RSV in the mouse. In the present studies, extensive pulmonary histopathology was observed in FI-RSV-immunized or RSV-infected BALB/c mice upon RSV challenge, and studies to determine the relative contributions of CD4+ or CD8+ ***T*** ***cells*** to this process were undertaken. Mice previously immunized with FI-RSV or infected with RSV were depleted of CD4+, CD8+, or both ***T*** - ***cell*** subsets immediately prior to RSV challenge, and the magnitude of inflammatory cell infiltration around bronchioles and pulmonary blood vessels and into alveolar spaces was quantified. The magnitude of infiltration at each anatomic site in previously FI-RSV-immunized or RSV-infected, nondepleted animals was similar, indicating that this is not a relevant model for enhanced disease. However, the effect of ***T*** - ***cell*** subset depletion on pulmonary histopathology following RSV challenge was very different between the two groups. Depletion of CD4+ ***T*** ***cells*** completely abrogated pulmonary histopathology in FI-RSV-immunized mice, whereas it had a much smaller effect on mice previously infected with RSV. FI-RSV-immunized or RSV-infected animals depleted of CD8+ ***T*** ***cells*** had only a modest reduction of pulmonary histopathology. In addition, RSV infection induced high levels of major histocompatibility complex class I-restricted cytotoxic ***T*** - ***cell*** activity, whereas FI-RSV ***immunization*** induced a low level. These data indicate that ***immunization*** with FI-RSV induces a cellular immune response different from that induced by RSV infection, which likely played a role in enhanced disease observed in infants and children.

CONTROLLED TERM: Check Tags: Animal; Comparative Study; Female
****Antibodies, Viral: AN, analysis***
*Antigens, CD4: IM, immunology
Formaldehyde
H-2 Antigens: IM, immunology
Haplotypes
Lung: IM, immunology
Lung: MI, microbiology
*Lung: PA, pathology
*Lymphocyte Depletion
Mice
Mice, Inbred BALB C
Mice, Inbred C57BL
*Respiratory Syncytial Viruses: IM, immunology
Respiratory Syncytial Viruses: PH, physiology
****Respiratory Syncytial Viruses: PY,*** pathogenicity***
Spleen: IM, immunology
*T-Lymphocyte Subsets: IM, immunology
****T-Lymphocytes, Cytotoxic: IM, immunology***
*Vaccines, Attenuated: IM, immunology
*Viral Vaccines: IM, immunology
Virus Replication
CAS REGISTRY NO.: 50-00-0 (Formaldehyde)
CHEMICAL NAME: 0 (***Antibodies*** , Viral); 0 (Antigens, CD4); 0

(H-2 Antigens); 0 (Vaccines, Attenuated); 0 (Viral Vaccines)

L30 ANSWER 11 OF 19 MEDLINE
ACCESSION NUMBER: 93037041 MEDLINE
TITLE: Distribution of immunocompetent cells in the endolymphatic sac.
AUTHOR: Kawauchi H; Ichimiya I; Kaneda N; Mogi G
CORPORATE SOURCE: Department of Otolaryngology, Medical College of Oita, Japan..
SOURCE: ANNALS OF OTOLOGY, RHINOLOGY, AND LARYNGOLOGY. SUPPLEMENT, (1992 Oct) 157 39-47.
Journal code: 5Q3. ISSN: 0096-8056.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 9301
ABSTRACT:
To better understand the role of immunocompetent cells in the defense mechanism of the inner ear, the distribution patterns of those cells were investigated in the endolymphatic sac (ES) of mice maintained in three different conditions: germ-free (GF), specific ***pathogen*** -free (SPF), and conventional (CV). In another experiment, the recruitment of lymphocyte subsets was examined in the ES of SPF rats undergoing a perilymphatic antigen challenge after systemic presensitization. In the ES of GF mice, no immunocompetent cells were found. In the ES of SPF and CV mice, cells positive for IgG, IgA, IgM, and Lyt-1 were present in much smaller numbers than in the ***nasal*** mucosa. Cells positive for Lyt-2 were not seen in the ES of any mice. In the ES of rats that underwent a perilymphatic antigenic stimulation after a systemic presensitization, ***B*** ***lymphocyte*** subsets (positive for IgG, IgA, IgM) were mobilized in increased numbers, and ***T*** ***cell*** subsets (helper/inducer and suppressor) were also found 1 week after perilymphatic antigen challenge. These results taken together suggest that the ES is not originally equipped to possess immunocompetent cells and mount an immune response, but that once it has been activated with the inner ear antigenic stimuli, the ES can be the active site of a local immune response of the inner ear.

CONTROLLED TERM: Check Tags: Animal; Male; Support, Non-U.S. Gov't
*** Antibody Formation***
Antigens: IM, immunology
*Endolymphatic Sac: IM, immunology
Enzyme-Linked Immunosorbent Assay
Germ-Free Life
Hemocyanin: IM, immunology
*** Immunization***
Immunoglobulins: AN, analysis
Immunohistochemistry
Lymphocyte Subsets
*Lymphocytes: IM, immunology
Mice
Mice, Inbred ICR
Perilymph: IM, immunology
Rats
Rats, Wistar
*** Specific Pathogen-Free Organisms***
CAS REGISTRY NO.: 9013-72-3 (Hemocyanin)
CHEMICAL NAME: 0 (keyhole-limpet hemocyanin); 0 (Antigens); 0 (Immunoglobulins)

L30 ANSWER 12 OF 19 MEDLINE
ACCESSION NUMBER: 88028394 MEDLINE
TITLE: Host defense impairments that may lead to respiratory infections.
AUTHOR: Reynolds H Y
CORPORATE SOURCE: Pulmonary Section, Yale University School of Medicine, New Haven, Connecticut..
SOURCE: CLINICS IN CHEST MEDICINE, (1987 Sep) 8 (3) 339-58.
Ref: 107
Journal code: DLR. ISSN: 0272-5231.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 8802
ABSTRACT:
Host defense mechanisms spaced along the respiratory tree and in the alveolar spaces effectively remove or contend with micro-organisms that enter the airways, so serious lung infections occur rarely in healthy people. Special circumstances, such as virgin exposure to a virulent microbe or a large innoculum of a ***pathogen*** , can result in illness, but usually routine surveillance host defenses are protective and suffice to keep colonizing airway flora in check. When pneumonia develops or recurrent sinopulmonary infection exists, however, some element of the normal defense apparatus may have failed or is inadequate. This review highlights several components of the apparatus, that is immunoglobulins IgG and IgA and the interaction of alveolar macrophages and lymphocytes, and examines deficiencies in their function that may result in infection. Along the conducting airways, poor mucociliary clearance and/or deficiencies in certain IgG subclass ***antibodies*** or destruction of IgA may predispose to sinopulmonary infections; these may be a manifestation of a hereditary disease. In pneumonia the alveolar macrophage is positioned as the central cell which must respond in several directions. This scavenger phagocyte first intercepts the microbe and either can kill or contain it or must call in some other phagocytic cell or inflammatory mediator(s) for assistance. Opsonic ***antibodies*** (IgG) and other nonimmune opsonins (complement and surfactant or fibronectin fragments) facilitate phagocytosis, but an absence of ***antibody*** may permit infection to develop with encapsulated bacteria (pneumococcus). Insufficient bone marrow reserves of PMNs or a paucity of chemotactic factors to attract them into the alveoli is a situation that may permit gram-negative bacilli and fungal organisms to flourish. Inability of immune ***T*** - ***lymphocytes*** to energize macrophages, through soluble cellular mediators that provide cell-mediated immunity and activation, makes containment of certain intracellular microbes impossible for these phagocytes (Legionella or mycobacteria). Likewise, concomitant infection of macrophages with viruses (human immunodeficiency virus, and

cytomegalovirus or herpes viruses) plus an excessive T-lymphocyte suppressor cell influence may make P. carinii and common bacterial and fungal organisms difficult to contain in the lungs of AIDS patients. Consideration about what the lung host deficiency might be can make therapy more specific through ***immunization*** to develop special ***antibodies*** , replacement of certain immunoglobulins (IgG subclasses), or selective ***administration*** of cell mediators (gamma-interferon or interleukins).

CONTROLLED TERM: Check Tags: Human
*** Antibody Formation***
Bronchoalveolar Lavage Fluid: CY, cytology
Bronchoalveolar Lavage Fluid: IM, immunology
Immunity, Cellular
Immunoglobulins: AN, analysis
*Immunologic Deficiency Syndromes: CO, complications
*** Nasal Mucosa: SE, secretion***
*Respiratory Tract Infections: ET, etiology
Respiratory Tract Infections: IM, immunology

L30 ANSWER 13 OF 19 MEDLINE
ACCESSION NUMBER: 75150165 MEDLINE
TITLE: The part played by cell-mediated immunity in mycoplasma respiratory infections.
AUTHOR: Taylor G; Taylor-Robinson D
SOURCE: DEVELOPMENTS IN BIOLOGICAL STANDARDIZATION, (1975) 28 195-210.
Journal code: E7V.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 7509
ABSTRACT:
Intranasal inoculation of M. pulmonis in mice and M. pneumoniae in hamsters results in pneumonia characterised by peribronchiolar and perivascular cuffing by lymphocytes. Thymus-dependent lymphocytes were depleted in mice by thymectomy and X-irradiation or treatment with anti-lymphocyte serum (ALS), and in hamsters by treatment with ALS. These procedures caused a reduction in the severity of pneumonic lesions in infected animals compared with infected immunologically normal animals. In addition, the organisms were present in slightly greater numbers in the lungs of the immunosuppressed animals. These results indicate the importance of thymus-dependent lymphocytes in the ***pathogenesis*** of mycoplasma-induced pulmonary disease. However, the role that these cells play in resistance to infection is not known and it may be that local secretory ***antibody*** is also important. Results of preliminary experiments involving hamster tracheal organ cultures infected with M. pneumoniae indicate that there is a factor present in lung washings from immune hamsters that protects against loss of ciliary activity brought about by M. pneumoniae.

CONTROLLED TERM: Check Tags: Animal; Comparative Study
*** Antibodies, Bacterial: AN, analysis***
Antigens, Bacterial
*** Antilymphocyte Serum: AD, administration &*** dosage***
Cilia: IM, immunology
Hamsters
*Immunity, Cellular
Immunosuppression
Injections, Intraperitoneal
Lung: IM, immunology
Lung: PA, pathology
Lymphocyte Depletion
Mice
*Mycoplasma Infections: IM, immunology
Mycoplasma Infections: PA, pathology
Radiation
*Respiratory Tract Infections: IM, immunology
Respiratory Tract Infections: PA, pathology
*** T-Lymphocytes: IM, immunology***
*** T-Lymphocytes: PH, physiology***
Thymectomy

L30 ANSWER 14 OF 19 SCISEARCH COPYRIGHT 1997 ISI (R)
ACCESSION NUMBER: 96:261509 SCISEARCH
THE GENUINE ARTICLE: UC314
TITLE: INDUCTION OF COMMON MUCOSAL IMMUNITY BY HORMONALLY IMMUNOMODULATED PERIPHERAL ***IMMUNIZATION***
AUTHOR: DAYNES R A (Reprint); ENIOUTINA E Y; BUTLER S; MU H H; MCGEE Z A; ARANEO B A
CORPORATE SOURCE: UNIV UTAH, SCH MED, DEPT PATHOL, SALT LAKE CITY, UT, 84132 (Reprint); UNIV UTAH, DEPT MED, DIV INFECT DIS, SALT LAKE CITY, UT, 84132; PARADIGM BIOSCI INC, SALT LAKE CITY, UT, 84132; VET AFFAIRS MED CTR, CTR GERIATR RES EDUC & CLIN, SALT LAKE CITY, UT, 84132
COUNTRY OF AUTHOR: USA
SOURCE: INFECTION AND IMMUNITY, (APR 1996) Vol. 64, No. 4, pp. 1100-1109.
ISSN: 0019-9567.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 53
ABSTRACT:
The study described in this report demonstrates that peripheral lymph nodes draining nonmucosal tissues can effectively serve as induction sites for the establishment of common mucosal immunity if the microenvironmental conditions are altered to mimic those normally present within mucosa-associated lymphoid tissues (e.g., Peyer's patches). Lymph node lymphocytes exposed in situ to the immunomodulatory influences of the hormone 1 alpha,25-dihydroxy vitamin D-3 were found to produce less gamma interferon and interleukin-2 (IL-2) and far more IL-4, IL-5, and IL-10 than lymphocytes from control animals. When coupled with vaccination with hepatitis B surface antigen (HBsAg), the hormone-immunomodulated switch from a peripheral lymph node phenotype to a Peyer's patch-like pattern promoted the induction of both a systemic and a common mucosal immune response. This was deter mined by the observed increased concentrations of serum anti-HBsAg ***antibody*** and by finding that anti-HBsAg secretory ***antibodies*** were detectable in urogenital, lachrymal, fecal, and oral secretions only in

the hormone-treated animals. In addition, specific ***antibody*** -secreting cells were detectable in the lamina propria of the lungs and small intestines of the hormone-treated animals subsequent to vaccination, indicating that the homing properties of antigen-specific ***B*** ***cells*** were being affected by the treatment procedure, The humoral and mucosal immune responses were further augmented if both 1 alpha,25-dihydroxy vitamin D-3 and dehydroepiandrosterone were used together as hormonal immunomodulators. This novel ***immunization*** technique may afford new opportunities to effectively intervene in sexually transmitted diseases and other diseases caused by mucosal ***pathogens*** .

CATEGORY: IMMUNOLOGY; INFECTIOUS DISEASES

SUPPL. TERM PLUS: HEPATITIS-B VIRUS; GROWTH-FACTOR-BETA; LYMPHOKINE PRODUCTION INVIVO; IMMUNOGLOBULIN-A; CHOLERA-TOXIN; ***T*** - ***CELLS*** ; HETEROSEXUAL TRANSMISSION; ***INTRANASAL*** ***IMMUNIZATION*** ; GAMMA-INTERFERON; SURFACE-ANTIGEN

RESEARCH FRONT: 94-7086 002; MUCOSAL IMMUNITY; ORAL IMMUNIZATION; HUMAN-IMMUNODEFICIENCY-VIRUS VACCINES; SECRETORY IGA; PNEUMOCOCCAL INFECTION; INDUCTION OF HUMORAL RESPONSES
94-0045 001; 1,25-DIHYDROXYVITAMIN D-3; BREAST-CANCER CELLS; CHOLECALCIFEROL ANALOGS
94-0261 001; CD40 LIGAND; HELPER T-CELL-DEPENDENT B-CELL ACTIVATION; DEFECTIVE EXPRESSION
94-2087 001; HEPATITIS-B VIRUS; Z-NUMBER-2 ALPHA(1)-ANTITRYPSIN TRANSGENIC MICE; RAF-DEPENDENT ACTIVATION OF C-JUN TRANSCRIPTIONAL ACTIVITY
94-5439 001; IL-2 RECEPTOR; X-LINKED SEVERE COMBINED IMMUNODEFICIENCY; GAMMA(C) CHAIN

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Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)
ABRAHAM E	1992	149	3719	J IMMUNOL
ALTER M J	1986	256	1307	JAMA-J AM MED ASSOC
ARANEO B A	1990		236	ADV GENE TECHNOLOGY
ARANEO B A	1991	78	688	BLOOD
ARANEO B A	1993	167	830	J INFECT DIS
ARCHIBALD D W	1990	3	954	J ACQ IMM DEF SYND
BEAGLEY K W	1992	21	347	GASTROENTEROL CLIN N
BEAGLEY K W	1988	141	2035	J IMMUNOL
BEASLEY R P	1981	2	1129	LANCET
BHALLA A K	1986	98	311	CELL IMMUNOL
DAVIES M D J	1981	22	481	GUT
DAYNES R A	1992	3	135	AGING IMMUNOL INFECT
DAYNES R A	1994	730	144	ANN NY ACAD SCI
DAYNES R A	1990	20	793	EUR J IMMUNOL
DAYNES R A	1990	171	979	J EXP MED
DAYNES R A	1991	174	1323	J EXP MED
DEFRANCE T	1992	175	671	J EXP MED
DEVOS T	1991	141	285	J IMMUNOL METHODS
FINKELMAN F D	1990	8	303	ANNU REV IMMUNOL
HAJISHENGALLIS G	1995	154	4322	J IMMUNOL
HARRIMAN G R	1988	140	3033	J IMMUNOL
HOLMGREN J	1993	11	1179	VACCINE
HOU M C	1993	167	938	J INFECT DIS
HUSBAND A J	1993	11	107	VACCINE
KILIAN M	1994		127	HDB MUCOSAL IMMUNOLO
KILSHAW P J	1990	20	2201	EUR J IMMUNOL
KIYONO H	1992	4	54	REG IMMUNOL
KRIESEL J D				IN PRESS J INFECT DI
LEBMAN D A	1990	144	952	J IMMUNOL
LEBMAN D A	1990	87	3962	P NATL ACAD SCI USA
LEMIRE J M	1994	135	2818	ENDOCRINOLOGY
LEMIRE J M	1992	49	26	J CELL BIOCHEM
LEMIRE J M	1991	87	1103	J CLIN INVEST
MAZANEC M B	1992	89	6901	P NATL ACAD SCI USA
MCGHEE J R	1992	10	75	VACCINE
MESTECKY J	1987	7	265	J CLIN IMMUNOL
MINGHETTI P P	1988	2	3043	FASEB J
MOSMANN T R	1989	7	145	ANNU REV IMMUNOL
MURRAY P D	1987	139	2669	J IMMUNOL
NEDRUD J G	1987	139	3484	J IMMUNOL
OBATA T	1991	10	574	BIOTECHNIQUES
PETKOVICH P M	1987	262	13424	J BIOL CHEM
REICHEL H	1987	84	3385	P NATL ACAD SCI USA
ROBERTS M	1993	11	866	VACCINE
ROMAGNANI S	1992	13	379	IMMUNOL TODAY
ROSENBLUM L S	1990	161	407	J INFECT DIS
SCHIEFERDECKER H L	1990	144	2541	J IMMUNOL
SCHUMACHER J H	1988	141	1576	J IMMUNOL
SEDGWICK J D	1986	87	37	J IMMUNOL METHODS
SVANBORG C	1994		71	HDB MUCOSAL IMMUNOLO
WEST D J	1989	17	172	AM J INFECT CONTROL
WILSON A D	1991	21	2333	EUR J IMMUNOL
WU H Y	1993	61	314	INFECT IMMUN

L30 ANSWER 15 OF 19 SCISEARCH COPYRIGHT 1997 ISI (R)

ACCESSION NUMBER: 95:504776 SCISEARCH

THE GENUINE ARTICLE: RK686

TITLE: INTRATRACHEAL GENE DELIVERY WITH ADENOVIRAL VECTOR INDUCES ELEVATED SYSTEMIC IGG AND MUCOSAL IGA ***ANTIBODIES*** TO ADENOVIRUS AND BETA-GALACTOSIDASE

AUTHOR: VANGINKEL F W; LIU C G; SIMECKA J W; DONG J Y; GREENWAY T; FRIZZELL R A; KIYONO H; MCGHEE J R; PASCUAL D W (Reprint)

CORPORATE SOURCE: UNIV ALABAMA, DEPT MICROBIOL, UNIV STN, BBRB 772, BIRMINGHAM, AL, 35294 (Reprint); UNIV ALABAMA, DEPT MICROBIOL, BIRMINGHAM, AL, 35294; UNIV ALABAMA, DEPT PHYSIOL & BIOPHYS, BIRMINGHAM, AL, 35294; UNIV ALABAMA, DEPT ORAL BIOL, BIRMINGHAM, AL, 35294; UNIV CALIF SAN FRANCISCO, DEPT LAB MED, SAN FRANCISCO, CA, 94143

COUNTRY OF AUTHOR: USA

SOURCE: HUMAN GENE THERAPY, (JUL 1995) Vol. 6, No. 7, pp. 895-903.
ISSN: 1043-0342.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 40
ABSTRACT:

One major concern about using adenoviral vectors for repetitive gene delivery to lung epithelial cells is the induction of an immune response to the vector, thus, impeding effective gene transduction. To assess the immune response to the adenoviral vector, repetitive intratracheal (i.t.) gene dosing was performed in CD-1 mice using the replication-deficient adenovirus 5 (Ade5) vector carrying the lacZ gene, and compared to the ***antibody*** responses induced by conventional ***intranasal*** (i.n.) and intraperitoneal (i.p.) routes of ***immunization*** . Kinetics of serum IgG, IgA, and IgM ***antibody*** responses to the adenoviral vector and to beta-galactosidase (beta-Gal) were evaluated. Two or three adenoviral vector doses given by i.t., i.n., or i.p. routes resulted in serum IgG titers in excess of 1:200,000, whereas serum IgM and IgA were moderately induced. Analysis of the predominant murine IgG subclass was determined to be IgG(2b) and IgG(2a). To determine the localization of this ***antibody*** response, the ELISPOT assay was employed. Lymphocytes were isolated from the lung, the lower respiratory lymph nodes (LRLN), the ***nasal*** passages (NP), and the spleen. For i.t- and i.n.-administered mice, the highest IgA spot-forming cell (SFC) response to Ade5 and beta-Gal was located in the NP and in the lung. Both the lung and the LRLN showed elevated numbers of IgG SFCs (4- to 12-fold greater than splenic IgG SFC response) for Ade5 and beta-Gal. This evidence suggests that the lung and associated lymphoid tissues were the source for serum ***antibodies*** . Further analysis of serum ***antibodies*** showed that the i.p.- and i.t.-administered groups yielded the greatest neutralization titers to Ade5, suggesting that the reduced effectiveness of repetitive gene transfer is in part due to circulating neutralizing ***antibodies*** . Thus, repetitive i.t. instillation will stimulate a localized and systemic ***antibody*** response to the vector.

CATEGORY: GENETICS & HEREDITY
SUPPL. TERM PLUS: CELL-SURFACE EXPRESSION; MURINE ***B*** - ***CELLS*** ; ENDOPLASMIC-RETICULUM; GLYCOPROTEIN-B; E3/19K PROTEIN; LYMPHOCYTES-T; SECRETION; ***PATHOGENESIS*** ; INFLUENZA; IMMUNITY
RESEARCH FRONT: 93-0868 003; CYSTIC-FIBROSIS TRANSMEMBRANE CONDUCTANCE REGULATOR; CFTR GENE; EFFECTS OF THE DELTA-F508 MUTATION
93-4539 001; ORAL IMMUNIZATION; COMMON MUCOSAL IMMUNE-SYSTEM; COW MILK IN SUCKLING RATS; INCREASED INVITRO INTESTINAL PERMEABILITY; IGA RESPONSES; T-CELL ACTIVATION
REFERENCE(S):

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)
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BERENCSI K	1993	74	2507	J GEN VIROL
BOTH G W	1993	193	940	VIROLOGY
BOUT A	1994	5	3	HUM GENE THER
BRODY S L	1994	5	821	HUM GENE THER
BURGERT H G	1987	6	2019	EMBO J
BURGERT H G	1987	84	1356	P NATL ACAD SCI USA
COX J H	1991	174	1629	J EXP MED
CZERKINSKY C C	1983	65	109	J IMMUNOL METHODS
DRUMM M L	1990	62	1227	CELL
ELDRIDGE J H	1991	28	287	MOL IMMUNOL
ENNIS F A	1982	58	273	J GEN VIROL
GALLICHAN W S	1993	168	622	J INFECT DIS
GINSBERG H S	1989	86	3823	P NATL ACAD SCI USA
GINSBERG H S	1991	88	1651	P NATL ACAD SCI USA
GRAHAM F L	1977	36	59	J GEN VIROL
GRAHAM F L	1992		363	VACCINES NEW APPROAC
KALTREIDER H B	1984		191	IMMUNOLOGY LUNG UPPE
MCGHEE J R	1993	2	55	INFECT AGENT DIS
MCINTYRE T M	1993	177	1031	J EXP MED
MOSMANN T R	1989	7	145	ANNU REV IMMUNOL
PASCUAL D W	1994	5	56	IMMUNOMETHODS
PASCUAL D W	1991	3	1223	INT IMMUNOL
PRINCE G A	1993	67	101	J VIROL
REYNOLDS H Y	1981	3	381	CURRENT PULMONOLOGY
RICH D P	1990	347	358	NATURE
ROSENFELD M A	1992	68	143	CELL
SIMECKA J W	1991	59	3715	INFECT IMMUN
SIMECKA J W	1992	4	18	REG IMMUNOL
SIMON R H	1993	4	771	HUM GENE THER
SNAPPER C M	1993	151	4625	J IMMUNOL
STRAUS S E	1984		451	ADENOVIRUSES
STREET N E	1991	5	171	FASEB J
TAYLOR P M	1985	54	607	IMMUNOLOGY
YAMADA Y K	1986	67	2325	J GEN VIROL
YANG Y	1994	91	4407	P NATL ACAD SCO IS
YEI S P	1994	1	192	GENE THER
YEI S P	1994	5	731	HUM GENE THER
ZABNER J	1993	75	1	CELL
ZABNER J	1994	6	75	NAT GENET

L30 ANSWER 16 OF 19 SCISEARCH COPYRIGHT 1997 ISI (R)
ACCESSION NUMBER: 92:62646 SCISEARCH
THE GENUINE ARTICLE: HA174
TITLE: INITIATION OF CYTOTOXIC ***T*** - ***CELL*** RESPONSE AND PROTECTION OF BALB/C MICE BY VACCINATION WITH AN EXPERIMENTAL ISCOMS RESPIRATORY SYNCYTIAL VIRUS SUBUNIT VACCINE
AUTHOR: TRUDEL M (Reprint); NADON F; SEGUIN C; BRAULT S; LUSIGNAN Y; LEMIEUX S
CORPORATE SOURCE: UNIV QUEBEC, INST ARMAND FRAPPIER, CTR RECH VIROL, 531 BLVD PRAIRIES, LAVAL H7V 1B7, QUEBEC, CANADA (Reprint); UNIV QUEBEC, INST ARMAND FRAPPIER, CTR RECH IMMUNOL, LAVAL H7V 1B7, QUEBEC, CANADA
COUNTRY OF AUTHOR: CANADA
SOURCE: VACCINE, (1992) Vol. 10, No. 2, pp. 107-112. ISSN: 0264-410X.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; AGRI
LANGUAGE: ENGLISH
REFERENCE COUNT: 35

ABSTRACT:

Respiratory syncytial virus is an important human ***pathogen*** causing serious lower respiratory tract infections of children and elderly people. Previous studies on the development of experimental subunit vaccines either expressed by recombinant DNA technology or prepared from purified viral proteins adsorbed on adjuvant (ISCOMs) have shown promise. The present work reports on the effectiveness of an experimental ISCOMs vaccine in initiating humoral and cell-mediated immune responses and in providing overall protection upon live virus challenge in Balb/c mice; results indicate that vaccination by the intramuscular route is more effective, even if vaccination by the ***intranasal*** route also significantly reduced virus shedding.

CATEGORY: IMMUNOLOGY

SUPPLEMENTARY TERM: RESPIRATORY SYNCYTIAL VIRUS; ISCOMS VACCINE; INTRAMUSCULAR; ***INTRANASAL*** ; ***T*** - ***CELL*** RESPONSE; HUMORAL RESPONSE

SUPPL. TERM PLUS: CHIMERIC FG GLYCOPROTEIN; COTTON RATS; MONOCLONAL-***ANTIBODIES*** ; MEDIATED-IMMUNITY; INFECTION; CHILDREN; RECOMBINANTS; ***IMMUNIZATION*** ; CHALLENGE; HAMSTERS

REFERENCE(S):

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)
ANDERSON L J	1985	151	626	J INFECT DIS
BANGHAM C R M	1986	137	3973	J IMMUNOL
BANGHAM C R M	1985	56	55	J VIROL
BELSHE R B	1982	145	311	J INFECT DIS
BRIDEAU R J	1989	70	2637	J GEN VIROL
COLLINS P L	1990	8	164	VACCINE
FISHAUT M	1980	96	179	J PEDIATR
FOSSUM C	1990	129	414	CELL IMMUNOL
FULGINITI V A	1968	89	435	AM J EPIDEMIOL
GIMENEZ H B	1986	67	863	J GEN VIROL
HEWLETT G	1989	117	243	J IMMUNOL METHODS
ISAACS D	1990	1	5	IMMUNOL INFECT DIS
ITO H	1986	56	125	JPN J EXP MED
JOHNSON P R	1987	61	3163	J VIROL
KAPIKIAN A Z	1969	89	405	AM J EPIDEMIOL
KIM H W	1968	89	422	AM J EPIDEMIOL
KIM H W	1976	10	75	PEDIATR RES
LEUNG K N	1982	67	312	CELL IMMUNOL
MOSSMANN T	1983	65	55	J IMMUNOL METHODS
MUFSON M A	1987	25	1535	J CLIN MICROBIOL
MUFSON M A	1985	66	2111	J GEN VIROL
MURPHY B R	1989	7	533	VACCINE
OLMSTED R A	1986	83	7462	P NATL ACAD SCI USA
PRINCE G A	1986	57	721	J VIROL
RAY R	1988	157	648	J INFECT DIS
STOTT E J	1986	60	607	J VIROL
TAYLOR G	1984	52	137	IMMUNOLOGY
TRUDEL M	1989		379	ADV MUCOSAL IMMUNOLO
TRUDEL M	1990	117	59	ARCH VIROL
TRUDEL M	1989	7	12	VACCINE
WALSH E E	1984	43	756	INFECT IMMUN
WATHEN M W	1989	70	2625	J GEN VIROL
WELLIVER R C	1988	39	147	ANNU REV MED
WRIGHT P F	1982	37	397	INFECT IMMUN
WRIGHT P F	1970	122	501	J INFECT DIS

L30 ANSWER 17 OF 19 EMBASE COPYRIGHT 1997 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 95313943 EMBASE

TITLE: Phenomenology, ***pathogenesis*** , diagnosis and treatment of aspirin-sensitive rhinosinusitis.

AUTHOR: Schapowal A.g.; Simon H.-U.; Schmitz-Schumann M.

CORPORATE SOURCE: Hochgebirgsklinik, 7265 Davos Wolfgang, Switzerland

SOURCE: Acta Oto-Rhino-Laryngologica Belgica, (1995) 49/3 (235-250).
ISSN: 0001-6497 CODEN: AORLAE

COUNTRY: Belgium

DOCUMENT TYPE: Journal

FILE SEGMENT: 005 General Pathology and Pathological Anatomy
011 Otorhinolaryngology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles

LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT:

Aspirin-sensitive rhinosinusitis is a non-allergic, non-infectious perennial eosinophilic rhinitis starting in middle age and rarely seen in children. It may also be seen in atopic patients who have developed a mixed type rhinitis with recurrent airway infections. There is an intolerance to aspirin and most other NSAID. An intolerance to tartrazine, food additives, alcohol, narcotics and local anaesthetics can follow. Most aspirin-sensitive patients developed ***nasal*** polyps. Untreated, it can lead to asthma. The frequency of aspirin intolerance is 6.18% in patients with perennial rhinitis and 14.68% in patients with ***nasal*** polyps. Immunologic studies of he blood and the ***nasal*** polyps show a hyperreactive immune system with an activation of the eosinophil granulocytes due to a TH1-lymphocyte-activation. In atopic subjects with a mixed type rhinitis, we found a TH2- and ***B*** - ***lymphocyte*** -activation as well. Inhibition of eosinophil apoptosis might be a second remarkable change in the immune system of aspirin-sensitive patients. A key ***pathogenic*** event for aspirin sensitivity is the change of the leukotriene pathway for arachidonic acid metabolism releasing high amounts of leukotrienes LTC4, LTD4 and LTE4, effective chemoattractants and activators of inflammatory cells. For the diagnosis of aspirin intolerance, ***nasal*** , bronchial and oral challenge are available. The sensitivity of ***nasal*** challenge with lysine-aspirin for the diagnosis of aspirin-sensitive rhinitis is 0.93, the specificity 0.97. It is the safest test in aspirin-sensitive asthmatics causing bronchial side effects only in 0.45%. Therapy of aspirin-sensitive rhinosinusitis includes avoidance of aspirin and NSAID. A general down regulation of the immune response with glucocorticosteroids is an effective means. We prefer a maintenance dose of budesonid 400 .mu.g a day. Systemic steroids for a reversibility test or in exacerbation due to viral infection are given in a dose of 50 mg a day for one week. If steroids don't work well enough, we combine them with aspirin desensitizations at a maintenance dose of 500 mg a day. Gastrointestinal side effects occur in 20% of the

patients with a dose of 500 mg aspirin a day, in 46% with a mean dose of 1300 mg a day. The combined treatment of topical ***nasal*** steroids and aspirin-desensitization is effective in 65% of the patients with improvement in the symptoms of hyper-secretion, irritation and blockage, while 73% show improvement of polyps, hyposmia and anosmia. Endonasal endoscopic surgery of the ethmoids, turbinectoms and septoplasty should be done if necessary, especially in cases were conservative treatment fails. After surgery a further antiinflammatory treatment is absolutely necessary otherwise polyps reoccur in 90% of the cases after weeks or months. Unfortunately there is so far no curative treatment. New drugs like cytokine or leukotriene receptor antagonists give hope for better results in treatment of aspirin intolerance in the future.

CONTROLLED TERM: EMTAGS: diagnosis (0140); etiology (0135); therapy (0160); adverse drug reaction (0198); iatrogenic disease (0300); mammal (0738); human (0888); ***intranasal drug administration*** (0283); article (0060)
Medical Descriptors:
*rhinitis: DI, diagnosis
*rhinitis: ET, etiology
*rhinitis: DT, drug therapy
*rhinitis: SU, surgery
*sinusitis: DI, diagnosis
*sinusitis: ET, etiology
*sinusitis: DT, drug therapy
*sinusitis: SU, surgery
drug hypersensitivity: SI, side effect
desensitization
urticaria: SI, side effect
asthma: SI, side effect
human
intranasal drug administration
article
Drug Descriptors:
*lysine acetylsalicylate: AE, adverse drug reaction
*acetylsalicylic acid: AE, adverse drug reaction
*nonsteroid antiinflammatory agent: AE, adverse drug reaction
*glucocorticoid: DT, drug therapy
*prednisone: DT, drug therapy
*budesonide: DT, drug therapy
tramadol: DT, drug therapy
leukotriene receptor blocking agent: DT, drug therapy
leukotriene receptor blocking agent: DV, drug development
antibody: DT, drug therapy
antibody: DV, drug development
CAS REGISTRY NO.: 34220-70-7; 37933-78-1; 62952-06-1; 77337-52-1; 50-78-2; 493-53-8; 53663-74-4; 53664-49-6; 63781-77-1; 53-03-2; 51333-22-3; 27203-92-5; 36282-47-0
CHEMICAL NAME: Tramal

L30 ANSWER 18 OF 19 EMBASE COPYRIGHT 1997 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 82202417 EMBASE

TITLE: Severe aplastic anaemia treated with anti-lymphocyte globulin. The relationship between clinical course and erythroid colony suppression by ***T*** ***cells*** .
AUTHOR: Hanada T.; Abe T.; Fukao K.; et al.
CORPORATE SOURCE: Inst. Clin. Med., Univ. Tsukuba, Sakura-Mura, Niihari-Gun, Ibaraki 305, Japan
SOURCE: SCAND. J. HAEMATOL., (1982) 29/2 (128-134).
CODEN: SJHAAQ
COUNTRY: Denmark
LANGUAGE: English
ABSTRACT:
A 6-year-old girl with severe aplastic anaemia improved promptly after treatment with anti-human lymphocyte globulin (ALG). Blood ***T*** ***lymphocytes*** were proved to have a suppressive effect on erythroid colony formation. ALG was administered intravenously at a dose of 15 mg/kg/d for 5 d. By the 14th d she showed a prompt increase in the reticulocyte count. Within the next 2 weeks slight increase of the platelet count was observed while the formation weakened when the reticulocyte count exceeded 100 x 109/l. About 4 months after ***administration*** of ALG, a decreased reticulocyte count was observed along with frequent ***nasal*** bleeding. Again the R cells exhibited a suppressive effect on erythroid colony formation. The results of serial co-culture studies revealed a close correlation between the ***T*** - ***cell*** suppressive effect on erythroid colony formation and reticulocyte response. The findings suggest an immune-mediated mechanism for the haematopoietic disorder in this patient.

CLASSIFICATION: 007.13.01.01.00.
007.16.00.00.00.
007.36.01.00.00.
025.01.03.00.00.
025.02.01.00.00.
025.04.04.00.00.
025.06.04.02.00.
026.03.04.00.00.
026.13.04.00.00.
026.14.01.00.00.
026.20.01.01.00.
037.24.05.00.00. Drug Literature Index/ANTISERA, TOXOIDS AND VACCINES/Allergens, antigens, antibodies
CONTROLLED TERM: EMTAGS: blood and hemopoietic system (0927); ***intravenous drug administration*** (0182); etiology (0135); immunological factors (0136); case report (0151); therapy (0160); mouse (0727); reticuloendothelial system (0924)
Medical Descriptors:
*aplastic anemia
*T lymphocyte
*suppressor cell
*erythropoiesis
*reticulocyte
****lymphocyte antibody***
medical treatment
pathogenesis
colony formation

L30 ANSWER 19 OF 19 EMBASE COPYRIGHT 1997 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 78260038 EMBASE
TITLE: Animal model of human disease: Infectious and neoplastic respiratory diseases associated with cigarette smoking.
AUTHOR: Holt P.G.; Keast D.; Mackenzie J.S.
CORPORATE SOURCE: Dept. Microbiol., Univ. West. Australia, Nedlands, Australia
SOURCE: AM. J. PATHOL., (1978) 90/1 (281-284).
CODEN: AJPAA4
COUNTRY: United States
LANGUAGE: English
ABSTRACT:
Mice exposed to the smoke of cigarettes exhibit biphasic changes in local and systemic immune function. ***Antibody*** production within the lung is severely depressed within 2 wk of starting exposure. In contrast, the regional lymph node and systemic activity show transient enhancement as long as 16 wk during continuous exposure prior to eventual suppression. Cellular immunity exhibits similar temporal changes. This biphasic phenomenon is also demonstrable in challenge experiments involving live influenza virus and viable tumor cells. These biphasic immunologic changes are also demonstrable following long-term exposure to industrial air pollutants. Therefore air pollution per se may induce respiratory disease(s). Likely, the agents in tobacco smoke which produce immunosuppression are chemically similar to industrial air pollutants (particularly nitrogen oxides), as many of the effects on animals produced by whole tobacco smoke can be mimicked by its vapor phase. Immunologic function in man is probably also affected by long-term inhalation of cigarette smoke. ***Antibody*** titers following ***immunization*** with killed influenza vaccines fall more rapidly in smokers than in nonsmokers, provided they have little or no immunity before vaccination; smokers have an increased susceptibility to influenza and other respiratory infections and seroconversion after ***intranasal*** ***administration*** of live influenza vaccine is increased; lymphocytotoxic ***antibody*** production against HLA antigens during consecutive pregnancies is less marked in smoking women than in nonsmokers; smokers consistently exhibit leukocytosis; alveolar macrophages from cigarette smokers fail to respond to the lymphokine MIF and chemotaxis is depressed in smokers. It has been suggested that a transient enhancement in some T-lymphocyte function similar to that observed in mouse may also occur in human smokers; PHA-reactivity and ***T*** - ***cell*** counts in the peripheral blood of smokers under 40 yr of age were above normal limits. Significantly, PHA-reactivity in smokers of 50 yr and above (together with circulating IgG levels) have been shown to be reduced. Cigarette smoke contains many carcinogens, which may induce lung cancer. Carcinogenic tobacco tars unlikely play a significant role in the etiology of infectious respiratory disease(s) in smokers, and consequently tobacco smoke components other than tars may also be deleterious to the health of the smoker. Similar immunologic mechanisms to those which provide the major defense against infectious disease(s), may protect the host from the development and spread of neoplasms. Prolonged cigarette smoking may, therefore, by immunosuppression, be involved in the etiology and ***pathogenesis*** of disease(s) associated with this habit.

CLASSIFICATION: 005.02.10.00.00.
005.02.13.00.00.
005.02.14.00.00.
005.02.19.00.00.
005.02.20.00.00.
005.03.10.03.00.
016.01.09.00.00.
016.02.01.03.00.
016.03.10.00.00.
CONTROLLED TERM: Medical Descriptors:
*smoking
*cigarette smoking
*cellular immunity
*lung carcinogenesis
*air pollution
*immunosuppression
*T lymphocyte
*lymphocyte transformation

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COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		30.33	30.48

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FILE 'USPAT' ENTERED AT 08:19:01 ON 31 JAN 97

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 *      WELCOME TO THE              *
 *    U.S. PATENT TEXT FILE         *
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=> s atomizer
L1 5380 ATOMIZER

=> s l1 and (nasal or intranasal)
7360 NASAL
1958 INTRANASAL
L2 166 L1 AND (NASAL OR INTRANASAL)

=> s l2 and device
898616 DEVICE
L3 81 L2 AND DEVICE

=> d 1

1. 5,594,987, Jan. 21, 1997, Method of making a sub-miniature aerosolizer; Theodore J. Century, 29/890.143, 456 [IMAGE AVAILABLE]

=> d 10

10. 5,561,149, Oct. 1, 1996, Use of certain imidazol carbazols in treating stress-related manic-depressive disorders; Moise Azria, et al., 514/397 [IMAGE AVAILABLE]

=> d20
'D20' IS NOT A RECOGNIZED COMMAND

=> d 20

20. 5,500,161, Mar. 19, 1996, Method for making hydrophobic polymeric microparticles; Alexander K. Andrianov, et al., 264/8, 9, 11, 12, 14 [IMAGE AVAILABLE]

=> d 30

30. 5,453,433, Sep. 26, 1995, Thiadiazoles and antipicornaviral compositions; David J. Aldous, et al., 514/362, 363; 548/127, 128, 129, 134, 135, 136, 142 [IMAGE AVAILABLE]

=> d 40

40. 5,112,868, May 12, 1992, Hydroxamate derivatives of selected nonsteroidal antiinflammatory acyl residues having cyclooxygenase and 5-lipoxygenase inhibition; Wiaczeslaw A. Cetenko, et al., 514/618, 255, 352, 371, 398, 426, 447, 472, 480, 561, 562, 563, 608, 617, 619, 620, 621, 622; 544/336; 546/309; 548/195, 346.1, 375.1, 557, 558; 560/9, 10, 11, 12, 13, 16, 20, 21, 22, 23, 24, 28, 29, 30, 31, 32, 33; 562/428, 434, 435, 437, 442, 443, 444, 448, 449, 450; 564/102, 162, 163, 164, 165, 166, 168, 169, 170, 172, 176, 180 [IMAGE AVAILABLE]

=> d 35

35. 5,234,932, Aug. 10, 1993, Substituted quinolines and leucotriene antagonism treatment therewith; Schneur Rachlin, et al., 514/311, 314; 546/172, 174, 175, 176, 180 [IMAGE AVAILABLE]

=> d 32

32. 5,401,777, Mar. 28, 1995, Use of preparations of curcuma plants; H. P. T. Ammon, et al., 514/731, 886, 925 [IMAGE AVAILABLE]

=> d 33-

33. 5,260,321, Nov. 9, 1993, Use of 1,4-dihydropyridine derivatives and combinations thereof with calcitonins; Robert P. Hof, et al., 514/338; 546/268.7, 269.1 [IMAGE AVAILABLE]

34. 5,250,293, Oct. 5, 1993, Method for the treatment of hypersensitivity diseases by administration of anionic polymers; Gerald J. Gleich, 424/78.04, 45, 78.02, 78.05, 441, 449, 456; 514/826, 887, 914 [IMAGE AVAILABLE]

35. 5,234,932, Aug. 10, 1993, Substituted quinolines and leucotriene antagonism treatment therewith; Schneur Rachlin, et al., 514/311, 314; 546/172, 174, 175, 176, 180 [IMAGE AVAILABLE]

36. 5,176,654, Jan. 5, 1993, Method and apparatus for otologic administration of medicament; Simeon B. Schreiber, 604/181, 187 [IMAGE AVAILABLE]

37. 5,173,274, Dec. 22, 1992, Flash liquid aerosol production method and appartus; Thomas E. Owen, 422/306; 239/3, 102.2; 252/305; 261/78.2, DIG.48 [IMAGE AVAILABLE]

38. 5,157,039, Oct. 20, 1992, 2-quinolinyl methoxy compounds, medical uses and intermediates therefor; Ole Bent T. Nielsen, et al., 514/311; 546/174, 176 [IMAGE AVAILABLE]

39. 5,155,110, Oct. 13, 1992, Fenamic acid hydroxamate derivatives having cyclooxygenase and 5-lipoxygenase inhibition; David T. Connor, et al., 514/258, 418, 507, 575; 544/285, 287; 548/483; 560/315; 562/622 [IMAGE AVAILABLE]

40. 5,112,868, May 12, 1992, Hydroxamate derivatives of selected nonsteroidal antiinflammatory acyl residues having cyclooxygenase and 5-lipoxygenase inhibition; Wiaczeslaw A. Cetenko, et al., 514/618, 255, 352, 371, 398, 426, 447, 472, 480, 561, 562, 563, 608, 617, 619, 620, 621, 622; 544/336; 546/309; 548/195, 346.1, 375.1, 557, 558; 560/9, 10, 11, 12, 13, 16, 20, 21, 22, 23, 24, 28, 29, 30, 31, 32, 33; 562/428, 434, 435, 437, 442, 443, 444, 448, 449, 450; 564/102, 162, 163, 164, 165, 166, 168, 169, 170, 172, 176, 180 [IMAGE AVAILABLE]

41. 5,112,804, May 12, 1992, Pharmaceutical composition and method of **intranasal** administration; Hanna R. Kowarski, 514/3; 424/434; 514/4, 12, 13, 14, 15, 947, 970 [IMAGE AVAILABLE]

42. 5,110,819, May 5, 1992, Substituted quinolines and medicinal use thereof; Ian Ahnfelt-Ronne, et al., 514/311, 232.5, 314; 544/128; 546/174, 176 [IMAGE AVAILABLE]

43. 5,109,009, Apr. 28, 1992, Quinoline and pyridine compounds and inhibition of 5-lipoxygenases therewith; Ole Bent T. Nielsen, et al., 514/311, 344, 345, 351; 546/172, 174, 177, 178, 179, 180, 286, 290 [IMAGE AVAILABLE]

44. 5,049,389, Sep. 17, 1991, Novel liposome composition for the treatment of interstitial lung diseases; Ramachandran Radhakrishnan, 424/450; 264/4.1; 424/434 [IMAGE AVAILABLE]

45. 5,043,165, Aug. 27, 1991, Novel liposome composition for sustained release of steroidal drugs; Ramachandran Radhakrishnan, 424/450; 514/180 [IMAGE AVAILABLE]

46. 5,038,769, Aug. 13, 1991, Method and apparatus for treating ailments; Robert S. Krauser, 128/203.27, 204.17 [IMAGE AVAILABLE]

47. 5,016,655, May 21, 1991, Cigarette manufacturing process; William J. Waddell, et al., 131/310, 331, 334, 335 [IMAGE AVAILABLE]

48. 4,967,772, Nov. 6, 1990, Tobacco smoking article and treatment of tobacco smoke with at least one alcohol; William J. Waddell, et al., 131/334, 31, 309, 310, 335, 337, 352 [IMAGE AVAILABLE]

49. 4,966,169, Oct. 30, 1990, Process for manufacturing cigarettes; William J. Waddell, et al., 131/31, 62, 79, 310, 335 [IMAGE AVAILABLE]

50. 4,945,098, Jul. 31, 1990, Novel propane-1,3-diol derivatives and use; Gunnar Grue-Sorensen, et al., 514/365, 247, 255, 256, 357, 358, 374, 399; 544/224, 335, 336; 546/145, 146, 147, 174, 175, 335, 341; 548/204, 236, 300.1, 338.1, 338.5, 341.5 [IMAGE AVAILABLE]

51. 4,943,587, Jul. 24, 1990, Hydroxamate derivatives of selected nonsteroidal antiinflammatory acyl residues and their use for cyclooxygenase and 5-lipoxygenase inhibition; Wiaczeslaw A. Cetenko, et al., 514/415, 233.2, 253; 544/143, 373; 548/495 [IMAGE AVAILABLE]

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53. 4,895,719, Jan. 23, 1990, Method and apparatus for administering dehydrated liposomes by inhalation; Ramachandran Radhakrishnan, et al., 424/45; 514/958, 959; 604/140 [IMAGE AVAILABLE]

54. 4,886,493, Dec. 12, 1989, Medical applicator process; Jordan Yee, 604/54, 49 [IMAGE AVAILABLE]

55. 4,876,283, Oct. 24, 1989, Antisnoring agent; Dietrich Reichert, 514/562, 655, 923 [IMAGE AVAILABLE]

56. 4,833,126, May 23, 1989, Pharmacologically active compounds and use; Geoffrey Allan, et al., 514/18, 826; 530/331 [IMAGE AVAILABLE]

57. 4,831,057, May 16, 1989, Antisnoring agent; Dietrich Reichert, 514/647 [IMAGE AVAILABLE]

58. 4,826,987, May 2, 1989, Pyridyl and quinoline derivatives; Ole Bent Iv rmose Nielsen, et al., 546/174, 22, 152, 170, 172, 176, 177, 286, 288, 289, 290, 296, 297, 298, 299, 300, 301, 302 [IMAGE AVAILABLE]

59. 4,805,609, Feb. 21, 1989, Pressurized ventilation system for patients; Josephine A. Roberts, et al., 128/200.21, 202.27, 203.12; 141/27; 239/338, 352 [IMAGE AVAILABLE]

60. 4,738,984, Apr. 19, 1988, Antirhinovirus agents; Roger A. Parker, 514/473 [IMAGE AVAILABLE]

61. 4,699,136, Oct. 13, 1987, Method and apparatus for treating ailments; Robert S. Krauser, 128/203.22, 203.27, 204.17 [IMAGE AVAILABLE]

62. 4,685,918, Aug. 11, 1987, Lipid osmotic pump; Gordon L. Amidon, et al., 424/467, 473; 604/892.1 [IMAGE AVAILABLE]

63. 4,670,419, Jun. 2, 1987, Pharmaceutical composition and its rectal use; Yoshiaki Uda, et al., 514/16, 17, 18, 19, 800, 806, 807, 809; 930/20, 21, 130 [IMAGE AVAILABLE]

64. 4,668,513, May 26, 1987, Method for combating snoring; Dietrich Reichert, 424/94.6, 94.1, 680; 514/458, 535, 642, 725, 758, 785 [IMAGE AVAILABLE]

65. 4,659,696, Apr. 21, 1987, Pharmaceutical composition and its **nasal** or vaginal use; Shin-ichiro Hirai, et al., 514/15, 16, 17, 18, 19; 930/20, 21, 130 [IMAGE AVAILABLE]

66. 4,602,099, Jul. 22, 1986, Antirhinovirus agents; Roger A. Parker, 549/479 [IMAGE AVAILABLE]

67. 4,556,557, Dec. 3, 1985, Method and composition for combating snoring, and the use of surface active substances for the preparation of said composition; Dietrich Reichert, 424/94.6, 94.63, 680; 514/458, 535, 642, 725, 758, 785 [IMAGE AVAILABLE]

68. 4,523,589, Jun. 18, 1985, Method and apparatus for treating ailments; Robert S. Krauser, 128/203.27, 204.17 [IMAGE AVAILABLE]

69. 4,498,485, Feb. 12, 1985, Interferon and interferon inducers combined with tobacco products; William A. Carter, 131/331, 310, 334, 335, 343, 352 [IMAGE AVAILABLE]

70. 4,442,112, Apr. 10, 1984, Dihydropyridine derivatives useful in treating vascular headaches; Else Muller-Schweinitzer, 514/338 [IMAGE AVAILABLE]

71. 4,300,546, Nov. 17, 1981, Hand-held **atomizer** especially for dispensing inhalation-administered medicaments; H. W. Kruber, 128/200.16; 222/199, 536, 537; 239/102.2 [IMAGE AVAILABLE]

72. 4,265,248, May 5, 1981, Differential olfactometer; Rene Chuiton, et al., 128/630, 747 [IMAGE AVAILABLE]

73. 4,228,795, Oct. 21, 1980, Apparatus for producing finely divided liquid spray; Robert S. Babington, 128/200.22; 239/338, 346 [IMAGE AVAILABLE]

74. 4,148,308, Apr. 10, 1979, Mouthpiece with a tongue retractor; William J. Sayer, 600/205; 128/207.14; 600/239 [IMAGE AVAILABLE]

75. 4,119,096, Oct. 10, 1978, Medical inhalation **device** for the treatment of diseases of the respiratory tract; Wolf-Dietrich Drews, 128/200.16; 239/102.2; 261/DIG.48, DIG.65 [IMAGE AVAILABLE]

76. 4,093,124, Jun. 6, 1978, **Atomizer** with air inlet valve; Bruno Morane, et al., 239/327; 128/200.14; 222/189.09 [IMAGE AVAILABLE]

77. 4,010,269, Mar. 1, 1977, Antiviral quinazoline compositions and methods of use; Harold E. Renis, et al., 514/260 [IMAGE AVAILABLE]

78. 3,971,377, Jul. 27, 1976, Medicament dispensing process for inhalation therapy; Nalinkant C. Damani, 128/200.17, 203.15, 203.21 [IMAGE AVAILABLE]

79. 3,949,743, Apr. 13, 1976, Medicated vapor production method and apparatus; Edward Shanbrom, 128/200.14, 203.17; 392/405; 607/84; D23/360; D24/110 [IMAGE AVAILABLE]

80. 3,834,682, Sep. 10, 1974, MIXING COLUMN FOR MEDICAL HUMIDIFIER AND METHOD OF HUMIDIFYING INHALABLE GASES; Charles J. McPhee, 261/123; 128/200.13; 261/DIG.65 [IMAGE AVAILABLE]

81. 3,831,606, Aug. 27, 1974, AUTO INHALER; Nalinkant C. Damani, 128/203.15 [IMAGE AVAILABLE]

=> d kwic 73

US PAT NO: 4,228,795 [IMAGE AVAILABLE] L3: 73 of 81

SUMMARY:

BSUM(3)

A **device** that disperses liquid into a fine spray or aerosol, used for medical purposes, is often called a nebulizer. When the. . .

SUMMARY:

BSUM(4)

Until . . . of atomization. While the ultrasonic nebulizer produces a superior aerosol as compared to conventional pneumatic systems, it is an expensive **device** using electronic components and thus may not be wholly dependable in service.

SUMMARY:

BSUM(13)

Devices . . . if the hand coordination of the user is not properly synchronized with his breathing rhythm. This type of aerosol spray **device** has also come under recent criticism by the U.S.F.D.A. because of the potential danger of using Freon propellants in therapeutic. . .

SUMMARY:

BSUM(15)

In still another type of **device**, the patient fills a small hand held nebulizer with medication, attaches the unit to a compressed gas source, and repeatedly. . .

DRAWING DESC:

DRWD(11)

FIG. 9 is a schematic elevational sectional view of a **device** suitable for nose inhalation.

DETDESC:

DETD(3)

In the embodiment of FIGS. 1 and 2, the **atomizer** includes an upper chamber 32 and a lower chamber 21, the two chambers being in communication via a lift tube. . .

DETDESC:

DETD(4)

Since the **atomizer** shown in FIGS. 1 and 2 has particular advantages as a medical nebulizer, it is illustrated with an impactor 26. . .

DETDESC:

DETD(8)

Optionally, the **atomizer** may also contain an air aspirator control means 31, affixed to the outlet of vent 20. When aspirator cap 31. . .

DETDESC:

DETD(10)

In the described operation, the **atomizer** will respond to the negative and positive pressures created by the respiratory rhythm of the patient and liquid will alternately. . .

DETDESC:

DETD(13)

If . . . could be placed in the nostril of a person, the embodiment shown in FIG. 1 could become a very effective **nasal** spray **device**. In this case the needed vacuum in upper chamber 32 would be created by inhalation through a patient's nose rather. . .

DETDESC:

DETD(27)

This . . . whose small moving parts are often prone to clogging. The FIG. 4 embodiment is also very well suited to a **nasal** mist sprayer because of the vertical spray pattern it produces. It is also contemplated that for such uses, outlet 46'. . .

DETDESC:

DETD(29)

FIGS. 5 and 6 illustrate a further embodiment of the present invention which is particularly adapted as a respiratory rhythm **atomizer**. The **device** includes a housing having upper and lower chambers 101 and 102 with partition 108 therebetween. Both the liquid to be. . .

DETDESC:

DETD(32)

Apparatus . . . used in the same manner as the apparatus shown in FIGS. 1 and 2. However, in using the earlier described **atomizer** shown in FIGS. 1 and 2, it is preferred that the patient simply inhale the desired amount of mist and. . .

DETDESC:

DETD(48)

FIG. 8 illustrates an embodiment especially designed as a continuous dual spray **atomizer** or nebulizer to produce two different aerosol streams. In the embodiments previously described, the spray produced is intermittent in nature,. . .

DETDESC:

DETD(53)

During . . . mist leaving upper discharge horn 315, is much finer than that leaving lower discharge horn 310, even though the same **atomizer** is supplying the mist that is eminating from both discharge horns. The liquid in the upper reservoir acts in a manner to filter the mist leaving said upper reservoir. In the steady-state operating mode of this spray **device**, the full liquid load is maintained in the upper reservoir, while a very small flow of liquid is supplied to. . .

=> d kwic 61

US PAT NO: 4,699,136 [IMAGE AVAILABLE] L3: 61 of 81

ABSTRACT:
A . . . of application where it is absorbed by the body. In the treatment of colds the air is introduced into the **nasal** passages of the cold sufferer at a hyperthermia level. A vaporized microbicidal agent is introduced into the stream of air and into the **nasal** passages. The apparatus includes a housing containing a fan or blower and temperature control heating elements to warm the air. The housing includes a distribution area having outlets for positioning on or about the **nasal** area of the user or other body area so that the warmed air is directed to flow to the desired. . . or other medicant within the apparatus housing is introduced into the flow stream of the heated air by a spray **device** so that minute droplets of the microbicidal agent or medicant are entrained within the flow stream of the heated air.. . .

SUMMARY:

BSUM(4)

Cold . . . climate of the blood and internal organs. The viruses attack the cells of the mucous membrane, producing congestion, sneezing and **nasal** drip. Some viruses have other effects, including aches, fever, coughing and chill. Colds take two to three days to incubate. . . peak. Sufferers are most infectious at the beginning, when sneezing and dripping are at their height. The virus kills the **nasal** cells it infects, and it takes time to regenerate them. That is one explanation of why it may take a. . .

SUMMARY:

BSUM(11)

Because it has been found that the cold virus exists predominately in the **nasal** passages where the temperature is lower, at 91.4.degree. F., than other body areas, it has been reasoned that by artificially heating the **nasal** passages above 98.6.degree. F., the cold virus might be killed or seriously weakened. The present invention applies earlier research into. . .

SUMMARY:

BSUM(13)

In . . . the present invention, original experiments utilized only the application of heat of approximately 100.degree. F. to 105.degree. F. to the **nasal** passages. These experiments showed that cold symptoms, while not lasting the full seven to ten day cycle, were only reduced. . . levels in combination with various microbicidal agents including hexylresorcinol and povidone-iodine. It was found that higher temperatures, which heated the **nasal** passages to approximately 106.degree. F. to 140.degree. F., in combination with the microbicidal agents hexylresorcinol and/or povidone-iodine resulted in cold. . .

SUMMARY:

BSUM(14)

It . . . the virus also acts as a catalyst to the body's immune system. The heat also increases the blood to the **nasal** passages, aiding in carrying away the dead cells and regenerating the new healthy cells in the **nasal** passages. Thus it was found that the treatment of early cold symptoms with heat and either hexylresorcinol or povidone-iodine, or. . .

SUMMARY:

BSUM(15)

For . . . to thirty-six hours. As used herein the term microbicidal agent means a germicide or antiseptic which, when applied in the **nasal** passages and used in conjunction with the application of heat to the **nasal** passages, produces an alleviation of cold symptoms in a cold sufferer. It is believed that the combination of the application. .

SUMMARY:

BSUM(26)

Various prior art devices have been proposed for the **device**, U.S. Pat. No. 1,239,634, produces a flow of warmed air to the patient but not at hyperthermia levels. However, the Stuart **device** will not be effective against colds as the amount of heat produced is virtually unregulated and not sufficiently high enough.. . . from the invention disclosed herein which produces controlled heated air to take advantage of the properties of hyperthermia. The Stuart **device**, using the disclosed filter, produces a very unmeasured amount of medicant as there is no way of controlling how much. . .

SUMMARY:

BSUM(27)

The Mascolo **device**, U.S. Pat. No. 1,965,424, utilizes steam passing through a closed cup of medicant. Again this **device** fails to present a means of controlling the temperature of the steam which is probably dangerously high, especially for children.. . . to ascertain the amount of thermal protection and the amount of medicant being delivered to the face and to the **nasal** passages, if any.

SUMMARY:

BSUM(28)

The Inoue **device**, U.S. Pat. No. 2,047,324, provides for the delivery of volatile matters or medicinal matters fumigated by means of an electric heating **device** and a forced draft. Again the **device** provides no control as to the amount of heat or the amount of medicant provided to the user.

SUMMARY:

BSUM(29)

The Conlin **device**, U.S. Pat. No. 3,522,236, provides a means of delivering vapors, perhaps medicated, to the user with a crude means of. . .

SUMMARY:

BSUM(30)

Specifically, . . . the safe treatment of the patient, and none disclose a way of stopping the medicant without shutting down the entire **device**.

SUMMARY:

BSUM(31)

Additionally, . . . advantages. These noted prior art devices are also bulky, barely portable and certainly not lightweight and handheld as is the **device** of the present invention with its obvious advantages particularly in treating another patient.

SUMMARY:

BSUM(33)

It . . . yet effective method and apparatus to treat symptoms of the common cold through the use of hyperthermia by warming the **nasal** passages of the cold sufferer and then providing for the application of a microbicidal agent within the **nasal** passages.

SUMMARY:

BSUM(34)

A . . . is to provide an apparatus for the treatment of the common cold which effectively combines the ability to heat the **nasal** passages of a cold sufferer to hyperthermic levels and to selectively deliver a microbicidal agent in convenient dosage to the warmed **nasal** passages.

SUMMARY:

BSUM(38)

Yet . . . provide an apparatus to deliver a variety of medicants for topical or internal use as a method of treatment. The **device** provides localized controlled and regulated hyperthermia as well as controlled and regulated medicant delivery.

SUMMARY:

BSUM(40)

The . . . cold sufferer wherein an air stream is heated to 110.degree. to 150.degree. F. The air stream is introduced into the **nasal** passage of the cold sufferer for a selected period of time and subsequently, an effective amount of sprays containing droplets of microbicidal and anti-viral agents, to apply a coating to the **nasal** passage lining, is also injected into the **nasal** passage in a timed concurrent or sequential relationship to the introduction of the heated air.

SUMMARY:

BSUM(41)

The . . . chosen microbicidal agents to combine in a synergistic manner to kill or seriously weaken the cold virus and/or bacteria. A **device** for carrying out the above method comprises a housing having air entry and air exit ports wherein air is drafted. . .

SUMMARY:

BSUM(42)

The . . . the exit port into the distribution area with the distribution area being adapted to distribute the heated air into the **nasal** passages of the cold sufferer.

SUMMARY:

BSUM(43)

The distribution area has appropriate **nasal** outlets directing the heated air and/or spray to the **nasal** passages. A fine spray of medicated droplets is selectively released to apply a medicated coating to the mucous lining of the **nasal** passages. The spray, having its own means of propulsion, may also be sprayed into the **nasal** passages independent of the forced heated air. Alternate means of providing the spray might be an attached **atomizer** bulb with a tube entering the housing or an electric piston pump instead of the mechanical pump. Alternatively, the air. . .

SUMMARY:

BSUM(44)

Another advantage of the present invention is that as a primarily dry heat **device** specific dosages of medicine can be delivered to the desired point without worrying about unmeasured dilution as might be caused. . .

SUMMARY:

BSUM(45)

Therefore, . . . of the present invention can be used for the delivery of medicants into the blood stream and body using the **nasal** or other mucosa and for the topical application of heat and medicine in the treatment of infection and diseases and. . .

SUMMARY:

BSUM(46)

For . . . mucous membranes act much faster and more effectively than pills or capsules ingested into the stomach and the method of **nasal** delivery is certainly more palatable than injection.

SUMMARY:

BSUM(48)

Use . . . replace much of the painful syringes and injections to which millions of people are subjected for delivery of medication. The **nasal** mucosa delivery method may also work for drugs unsuitable for the new skin patches and also drugs currently being delivered. . .

SUMMARY:

BSUM(53)

Among . . . and punctures, boils, warts and other skin growths, and the treatment of allergic rhinitis and sore throats among others. The **device** is also usable to deliver medicants at hyperthermia levels to the anal passage and can effectively replace medication normally administered. . .

SUMMARY:

BSUM(54)

Colds, . . . with a broad spectrum antiseptic such as hexylresorcinol in an aqueous solution, which are directed in controlled amounts to the **nasal** passages. The medicant is applied intermittently during the heat treatment. The hyperthermia kills or weakens the virus and bacteria in. . .

DRAWING DESC:

DRWD(3)

FIG. 2 is a vertical cross-section diagramatically illustrating the **device** of the present invention;

DETDESC:

DETD(2)

With . . . filter out dust particles. Preferably housings 12 and 14 are formed of lightweight high strength molded plastic material so the **device** is readily adapted for ready portability and ease of use.

DETDESC:

DETD(3)

Mounted . . . to draw atmospheric air through opening 20 and screen 24 into chamber 16. The air flow passes through a heating **device** 28 and continues under the action of blower 26 after being warmed by the heater 28 to exit opening 22.

DETDESC:

DETD(4)

Housing . . . are designed to direct the flow of air from the apparatus 10 to the nostril of a user of the **device**. To this end the snap on end pad 34 is preferably made of a somewhat pliable rubber-like material for comfort and convenience in use. As illustrated in FIG. 2, the nose 44 of an intended user of the **device** may be pressed against end wall 36. This action tends to elevate the position of the nostrils, indicated at 46 to locate the nostrils of a user of the **device** in convenient position over the air exits ports 40 and 42.

DETDESC:

DETD(6)

The temperature of the air warmed by heater 28 may be conveniently controlled by a variable **device** such as a rheostat 50. Adjustment of rheostat 50 varies the current flowing to the heater element 28 thereby controlling. . .

DETDESC:

DETD(7)

Mounted . . . an area of chamber 16 adjacent the outlet ports 40 and 42 for convenient inhalation by a user of the **device**. The air flowing under action of blower 26 entrains the minute droplets of the microbicidal agent to assist in the. . .

DETDESC:

DETD(8)

The spray **device** 58 may also be operated independently of blower actuation thus affording use of the **device** as an inhalation **device** without the flow of warmed heated air. The **device** may also be operated with the blower and spray alone without activating the heater element 28 to assist in the. . .

DETDESC:

DETD(10)

Because . . . need not be of overly large capacity and the blower need only direct a relatively modest flow of air, the **device** is advantageously of a relatively compact and lightweight construction facilitating convenient handheld use.

DETDESC:

DETD(12)

In . . . 4, delivery nozzles 110 are appropriately affixed, if desired, to the outlet ports 40, 42 for extension into the user's **nasal** passages. Nozzles 110 each are cylindrical members having a rounded exterior segment 112 provided with an access port 114 to provide fluid communication to the interior of the **device**. Nozzles 110 may be formed integral with pad 34 or as insertable members therein. It is, of course, recognized that a suitably shaped anal delivery nozzle may also be employed when the **device** is used to administer medication to the anal passages and that other suitable shapes may be employed for delivery of. . .

DETDESC:

DETD(13)

In . . . was applied to warm the air to between 110.degree. F. to 130.degree. F. to induce an elevated temperature within the **nasal** passages of the user to a hyperthermia level, i.e. above about 106.degree. F. The heated air was supplied at intermittent periods for comfort and periodically spray mists of the hexylresorcinol in sufficient amounts to effectively coat the **nasal** passages were induced to enter the warmed **nasal** passages.

DETDESC:

DETD(14)

One recommended procedure is the introduction of the microbicidal spray into the **nasal** passage in a timed relationship to the introduction of the heated air stream and forms a sequence of operation which. . .

DETDESC:

DETD(48)

In addition the **device** may be used for the application of medicants in a topical manner on other parts of the body. As an. . .

DETDESC:

DETD(49)

Further, the **device** of the present invention has been found to be effective to heat and dry the area between the toes and. . .

CLAIMS:

CLMS(5)

5. Apparatus as defined in claim 1 wherein said **device** is constructed of lightweight material for convenient handheld use.

=> log y
U.S. Patent & Trademark Office LOGOFF AT 08:28:36 ON 31 JAN 97

FILE 'USPAT' ENTERED AT 08:51:35 ON 31 JAN 97

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 ********************************
 *       WELCOME TO THE         *
 *    U.S. PATENT TEXT FILE     *
 ********************************

=> s anti-human cd4
     135266 ANTI
     141624 HUMAN
       1213 CD4
L1        11 ANTI-HUMAN CD4
             (ANTI(W)HUMAN(W)CD4)

=> s l1 and hybridoma
       3409 HYBRIDOMA
L2         7 L1 AND HYBRIDOMA

=> d 1-
```

1. 5,594,120, Jan. 14, 1997, Integrin alpha subunit; Michael B. Brenner, et al., 536/23.5; 435/172.3, 240.2, 320.1; 536/24.31, 24.33 [IMAGE AVAILABLE]

2. 5,583,002, Dec. 10, 1996, Evaluation and treatment of patients with progressive immunosuppression; Augusto C. Ochoa, et al., 435/7.23; 424/9.2, 93.71; 435/7.24, 7.4, 15, 29; 436/63, 64, 86, 501 [IMAGE AVAILABLE]

3. 5,556,763, Sep. 17, 1996, Evaluation and treatment of patients with progressive immunosuppression; Augusto C. Ochoa, et al., 435/7.23; 424/9.2, 93.71; 435/6, 7.24; 436/501 [IMAGE AVAILABLE]

4. 5,525,461, Jun. 11, 1996, Therapeutic and diagnostic methods using total leukocyte surface antigens; Charles W. Rittershaus, 435/5, 7.1, 7.2, 7.21, 7.22, 7.23, 7.24, 7.92, 7.93, 7.94, 7.95, 974, 975 [IMAGE AVAILABLE]

5. 5,518,882, May 21, 1996, Immunological methods of component selection and recovery; Garry Lund, et al., 435/6, 7.21, 7.5, 7.8, 7.93; 436/501, 518, 541, 543 [IMAGE AVAILABLE]

6. 5,426,029, Jun. 20, 1995, Therapeutic and diagnostic methods using leukocyte surface antigens; Charles W. Rittershaus, et al., 435/7.21, 7.24, 7.9, 7.94; 436/501, 506, 518, 536 [IMAGE AVAILABLE]

7. 5,292,636, Mar. 8, 1994, Therapeutic and diagnostic methods using soluble T cell surface molecules; Patrick C. Kung, et al., 435/5, 7.23, 7.24, 7.9, 7.94, 34, 974, 975; 436/506, 518, 536, 548, 811, 813 [IMAGE AVAILABLE]

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=> d kwic 7
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US PAT NO: 5,292,636 [IMAGE AVAILABLE] L2: 7 of 7

SUMMARY:

BSUM(165)

. . .

T 76:4061-4065;
cells) Ledbetter, J. A., et al., 1981 Monoclonal Antibodies and T Cell **Hybridoma** Elsevier/ North Holland, N.Y., pp. 16-22.
T6 49 Thymocytes & OKT6 Reinherz, 1979, Langerhans NAI/34. . .

DETDESC:

DETD(33)

A . . . for the production of antibody molecules by continuous cell lines in culture. These include but are not limited to the **hybridoma** technique originally described by Kohler and Milstein (1975, Nature 256:495-497), and the more recent human B cell **hybridoma** technique (Kozbor et al., 1983, Immunology Today 4:72) and EBV-**hybridoma** technique (Cole et al., 1985, Monoclonal Antibodies and Cancer Therapy, Alan R. Liss, Inc., pp. 77-96).

DETDESC:

DETD(54)

Monoclonal . . . were produced as previously described (Uchiyama, T., et al., 1981, J. Immunol. 126(4) 1393-1397; Rubin, L. A., et al., 1985, **Hybridoma** 4:91-102; Jung, L. K. L., et al., 1984, J. Exp. Med. 160:1957). Additionally, monoclonal antibodies directed against IL2R may be. . .

DETDESC:

DETD(290)

Mouse monoclonal antibodies were generated according to procedure as described (Rubin, L. A., et al., 1985, **Hybridoma** 4:91-102; Kohler, G. and Milstein, C., 1975, Nature 256:495-497). The two monoclonal antibodies selected (2R12, 7G7) are directed against different. . .

DETDESC:

DETD(312)

Both . . . and sequential immunoprecipitations demonstrate that this molecule is identical to that precipitated by anti-Tac (Rubin, L. A., et al., 1985, **Hybridoma** 4:91-102). Thus, one of the antibodies (2R12) demonstrates competitive binding with anti-Tac in cytofluorometric analysis of activated lymphocytes. The enzyme. . .

DETDESC:

DETD(518)

Antibodies . . . and incubated with recombinant soluble CD4 for 2 hours at 37.degree. C. Plates were washed and 50 .mu.l of each **hybridoma** supernatant at 1-10 .mu.g/ml were added followed by 50 .mu.l of biotinyl Leu3A. Following a 2 hour incubation, plates were. .

DETDESC:

DETD(521)

The . . . of a microtiter plate (Nunc, certified high binding) was coated overnight at 4.degree. C. with a solution of murine monoclonal **anti**-**human** **CD4** antibody in PBS, pH 7.4. Any remaining protein-binding sites on the microtiter wells were then blocked for two hours at. . . three times with 350 .mu.l of PBS/Tween 20 as above. One hundred .mu.l of horseradish peroxidase (HRP) conjugated murine monoclonal **anti**-**human** **CD4** antibody was added to each well of the microtiter plate, and the plate was again incubated at 37.degree. C. for. . .

DETDESC:

DETD(543)

Antibodies . . . from a mouse immunized with whole T cells and screened for their ability to replace Leu3a in an assay. 500 **hybridoma** clones were screened and three clones meeting the above criteria were identified. One of these clones, termed 8F4, showed the. .

DETDESC:

DETD(689)

The following **hybridoma** cell lines, producing the indicated monoclonal antibody, have been deposited with the American Type Culture Collection, Rockville, Md., and have. . .

DETDESC:

DETD(690)

Hybridoma	Accession Monoclonal Antibody Number
Cell line AM92/2R12	
AM92/2R12 (anti-IL2R)	HB 9341
Cell line 7G7 7G7 (anti-IL2R)	HB. . .

CLAIMS:

CLMS(5)

5. . . . according to claim 1, 2 or 4 in which the first antibody comprises monoclonal antibody 8F4 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9843.

CLAIMS:

CLMS(6)

6. . . . according to claim 1, 2 or 4 in which the second antibody comprises monoclonal antibody R2B7 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9842.

CLAIMS:

CLMS(7)

7. . . . according to claim 1, 2 or 4 in which the first antibody comprises monoclonal antibody 8F4 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9843, and the second antibody comprises monoclonal antibody R2B7 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9842.

CLAIMS:

CLMS(8)

8. . . . in which the first antibody has the same epitope specificity as that of monoclonal antibody 8F4 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9843.

CLAIMS:

CLMS(9)

9. . . . in which the second antibody has the same epitope specificity as that of monoclonal antibody R2B7 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9842.

CLAIMS:

CLMS(10)

10. . . . in which the first antibody has the same epitope specificity as that of monoclonal antibody 8F4 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9843, and the second antibody has the same epitope specificity as that of monoclonal antibody R2B7 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9842.

CLAIMS:

CLMS(16)

16. The kit of claim 12 in which the first antibody comprises monoclonal antibody 8F4 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9843.

CLAIMS:

CLMS(17)

17. The kit of claim 12 in which the second antibody comprises monoclonal antibody R2B7 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9842.

CLAIMS:

CLMS(18)

18. The kit of claim 12 in which the first antibody comprises monoclonal antibody 8F4 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9843, and the second antibody comprises monoclonal antibody R2B7 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9842.

CLAIMS:

CLMS(23)

23. . . . in which the first antibody has the same epitope specificity as that of monoclonal antibody 8F4 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9843.

CLAIMS:

CLMS(24)

24. . . . in which the second antibody has the same epitope specificity as that of monoclonal antibody R2B7 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9842.

CLAIMS:

CLMS(25)

25. . . . in which the first antibody has the same epitope specificity as that of monoclonal antibody 8F4 as produced by the **hybridoma** deposited with the ATCC and assigned accession number HB 9843, and the second antibody has the same epitope specificity as that of monoclonal antibody R2B7 as produced by he **hybridoma** deposited with the ATCC and assigned accession number HB 9842.

=> d kwic 6

US PAT NO: 5,426,029 [IMAGE AVAILABLE] L2: 6 of 7

SUMMARY:

BSUM(10)
2. Henry et al., 1989, **Hybridoma**, 8:577.

SUMMARY:

BSUM(22)
14. Ledbetter et al., 1981, Monoclonal Antibodies and T cell **Hybridoma**, Elsevier, North Holland, N.Y. pp 16-22.

SUMMARY:

BSUM(82)

.beta.FI . . . .beta. chain of the .alpha..beta. TCR and identifies all T cells expressing the .alpha..beta. TCR. .alpha.F1 (Henry et al., 1989, **Hybridoma** 8:577) is a monoclonal antibody specific for a framework determinant of the .alpha. chain and identifies all T cells expressing. . .

DETDESC:

DETD(47)

A . . . for the production of antibody molecules by continuous cell lines in culture. These include but are not limited to the **hybridoma** technique originally described by Kohler and Milstein (1975, Nature 256:495-497), and the more recent human B cell **hybridoma** technique (Kozbor et al., 1983, Immunology Today 4:72) and EBV-**hybridoma** technique (Cole et al., 1985, Monoclonal Antibodies and Cancer Therapy, Alan R. Liss, Inc., pp. 77-96).

DETDESC:

DETD(116)

Antibodies . . . and incubated with recombinant soluble CD4 for 2 hours at 37.degree. C. Plates were washed and 50 .mu.l of each **hybridoma** supernatant at 1-10 .mu.g/ml were added followed by 50 .mu.l of biotinyl Leu3A. Following a 2 hour incubation, plates were. .

DETDESC:

DETD(119)

The . . . of a microtiter plate (Nunc, certified high binding) was coated overnight at 4.degree. C. with a solution of murine monoclonal **anti**-**human** **CD4** antibody in PBS, pH 7.4. Any remaining protein-binding sites on the microtiter wells were then blocked for two hours at. . . three times with 350 .mu.l of PBS/Tween 20 as above. One hundred .mu.l of horseradish peroxidase (HRP) conjugated murine

monoclonal **anti**-**human** **CD4** antibody was added to each well of the microtiter plate, and the plate was again incubated at 37.degree. C. for. . .

DETDESC:

DETD(141)

Antibodies . . . from a mouse immunized with whole T cells and screened for their ability to replace Leu3a in an assay. 500 **hybridoma** clones were screened and three clones meeting the above criteria were identified. One of these clones, termed 8F4, showed the. .

DETDESC:

DETD(192)

Total . . . Tween 20). After aspirating the final wash buffer from the wells, 50 .mu.l of horseradish peroxidase (HRP) conjugated murine monoclonal **anti**-**human** **CD4** antibody (in PBS with 15% FCS and 0.15% NP-40) and 50 .mu.l of sample or standard were added to each. . .

DETDESC:

DETD(353)

The following **hybridoma** cell lines, producing the indicated monoclonal antibody, have been deposited with the American Type Culture Collection, Rockville, Md., and have. . .

DETDESC:

DETD(354)

Hybridoma	Accession Monoclonal Antibody Number
Cell line AM92/2R12	
AM92/2R12 (anti-IL2R)	HB 9341
Cell line 7G7 7G7 (anti-IL2R) HB. . .	

=> s human antigen presenting cells
141624 HUMAN
15216 ANTIGEN
50175 PRESENTING
143167 CELLS
L3 1 HUMAN ANTIGEN PRESENTING CELLS
(HUMAN(W)ANTIGEN(W)PRESENTING(W)CELLS)

=> d

1. 5,476,996, Dec. 19, 1995, Human immune system in non-human animal; Darcy B. Wilson, et al., 800/2; 424/9.1, 93.1, 93.7, 93.71, 534, 577, 578; 800/DIG.2, DIG.5 [IMAGE AVAILABLE]

=> s HLA
L4 938 HLA

=> s l4 and class 2
124019 CLASS
2111984 2
1293 CLASS 2
(CLASS(W)2)
L5 10 L4 AND CLASS 2

=> d 1-

1. 5,550,214, Aug. 27, 1996, Isolated antigenic oncogene peptide fragments and uses; Timothy J. Eberlein, et al., 530/328; 424/154.1, 155.1, 174.1, 184.1, 185.1, 277.1; 530/300, 403; 930/230 [IMAGE AVAILABLE]

2. 5,503,976, Apr. 2, 1996, DNA sequences coding for the DR .beta.-chain locus of the human lymphocyte antigen complex and diagnostic typing processes and products related thereto; Bernard F. Mach, et al., 435/6; 536/23.5 [IMAGE AVAILABLE]

3. 5,468,612, Nov. 21, 1995, 9804 gene and methods of use thereof; Edward H. Cohen, et al., 435/6, 240.2, 320.1; 536/23.5; 935/1, 22, 24, 76, 77, 78 [IMAGE AVAILABLE]

4. 5,426,181, Jun. 20, 1995, DNA encoding cytokine-induced protein, TSG-14; Tae H. Lee, et al., 536/23.5; 435/69.1, 252.3, 320.1; 536/23.1 [IMAGE AVAILABLE]

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US PAT NO: 5,006,470 [IMAGE AVAILABLE] L5: 8 of 10

ABSTRACT:
This . . . invention provides a human monoclonal antibody, produced by a hybridoma cell line designated DSM1, which specifically binds to a human **class** **2** tumor protein antigen.

SUMMARY:

BSUM(4)

Serological . . . 1 (unique) melanoma antigens are restricted to the autologous melanoma; six examples of class 1 antigens have been detected (10-15). **Class** **2** melanoma antigens are detected on the autologous melanoma, on a subset of allogeneic melanoma cells, and on other neuroectodermally derived tumors; these **class** **2** antigens have characteristics of autoimmunogenic differentiation antigens, and one of the best-analyzed **class** **2** melanoma antigens is the ganaglioside GD2 (16). Class 3 melanoma antigens are not restricted to any differentiation lineage and are. . .

SUMMARY:

BSUM(11)

This invention also provides for a human monoclonal antibody which specifically binds to a human **class** **2** tumor protein antigen. Additionally, this invention provides for a hybridoma cell line designated DSM1 which produces the human monoclonal antibody.

DETDESC:

DETD(5)

This invention still further provides a human monoclonal antibody which specifically binds to a human **class** **2** tumor protein antigen. Additionally, this invention provides for a hybridoma cell line designated DSM1 which produces the human monoclonal antibody.

DETDESC:

DETD(28)

DSM1 . . . the SK-MEL-13 target cell (AH) did not absorb reactivity from DSM1 or DS serum, suggesting that alloantigenic systems such as **HLA**, class 1, or **class** **2** antigens were not involved. The antigen detected by DSM1 is heat-labile, hydrophobic, and binds to Con A.

DETDESC:

DETD(30)

Several . . . antigen will facilitate the study of this class of antigen. The antigen detected by HJM1 has the characteristics of a **class** **2** melanoma antigen, i.e., expression by the autologous melanoma, and a subset of allogeneic melanomas, but not by cells of nonneuroectodermal. . .

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US PAT NO: 4,693,966 [IMAGE AVAILABLE] L5: 9 of 10

SUMMARY:

BSUM(3)

The . . . hepatoma (Giraldo, G. and E. Beth. 1980, The Role of Viruses in Human Cancer. Vol. 1 (Elsevier/North-Holland, New York)), and **HLA** antigens and blood group antigens, the nature and significance of other classes of human cancer antigens detected by human antibody. . . Class 1 antigens are restricted to autologous tumor cells, not being detected on any other cell type, normal or malignant. **Class** **2** antigens are shared antigens, found on a proportion of allogeneic tumors as well as on autologous tumors; recent evidence indicates that some **Class** **2** antigens are autoantigenic differentiation antigens, as they are detected on a restricted range of normal tissues (Watanabe, T., C. S.. . . broadly represented antigens have not been extensively analyzed. Whereas Class 3 reactivity is relatively common, antibodies to Class 1 and **Class** **2** antigens are found infrequently (10% of patients).

=> s cd21
L6 25 CD21

=> s l6 and human
141624 HUMAN
L7 21 L6 AND HUMAN

=> s l7 and antibod?
21935 ANTIBOD?
L8 20 L7 AND ANTIBOD?

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3. 5,583,033, Dec. 10, 1996, T lymphocyte precursor; Leon W. M. M. Terstappen, et al., 435/240.2, 7.21, 7.24 [IMAGE AVAILABLE]

4. 5,580,756, Dec. 3, 1996, B7IG fusion protein; Peter S. Linsley, et al., 435/69.7, 91.1; 530/350, 387.1, 387.3, 395; 536/23.4 [IMAGE AVAILABLE]

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10. 5,474,771, Dec. 12, 1995, Murine monoclonal **antibody** (5c8) recognizes a **human** glycoprotein on the surface of T-lymphocytes, compositions containing same; Seth Lederman, et al., 424/133.1, 130.1, 144.1, 153.1, 154.1; 435/70.21, 172.2, 240.27; 530/388.7, 388.73, 388.75 [IMAGE AVAILABLE]

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TITLE: Murine monoclonal **antibody** (5c8) recognizes a **human** glycoprotein on the surface of T-lymphocytes, compositions containing same

US PAT NO: 5,474,771 DATE ISSUED: Dec. 12, 1995
[IMAGE AVAILABLE]

APPL-NO: 07/792,728 DATE FILED: Nov. 15, 1991

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